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THE RESULTS OF SECONDARY SUTURE OF PERIPHERAL NERVES.[1]

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I.—Introduction.

Although further time must elapse before the end-results of secondary suture of peripheral nerves, injured in the war, can be judged in large numbers, it is now possible to communicate a preliminary report upon the progress to date of those patients in which this method of surgical treatment has been found necessary; and it seems advisable to do this for the benefit of the many pensioners who still await further treatment.

So many factors may influence the progress and extent of recovery that it is exceedingly difficult to interpret all the observations, or estimate the influence of any one individual factor. To attempt the elucidation of the numerous problems encountered, or to provide any guidance for the future, it is obviously necessary to consider large numbers of patients on whom regular systematic examinations have been made and careful records kept of all complications, methods of

[1] A Report to the Medical Research Council.

treatment, and progress. No trustworthy conclusions can be drawn from the publication of a few isolated cases, or even from a large series, unless attention has been directed to all the factors which may prevent or cause delay or acceleration of recovery. The closest co-operation between the surgeon and the investigator is essential; if the latter is unable to be present at the operation, it is of fundamental importance that the most detailed notes should be provided of the anatomical and pathological conditions found on exposure of the nerve, together with an exact account of the surgical procedure adopted. It has been my good fortune to have the fullest possible support from my surgical colleagues at the Grangethorpe Hospital, Manchester, and I would like to express my deep appreciation of the careful and elaborate system of note-taking which they have instituted, as, without the information supplied by them, it would have been impossible to attempt an inquiry into the problems of secondary suture.

In this report are included all the results of secondary suture, where it has been possible to continue observations for a period of not less than six months after the date of the operation. It was rarely possible to follow the patients even for so short a time before 1917, when the special hospitals were instituted; consequently almost all the patients quoted have been operated upon since the beginning of that year, and only a small number can be considered as end-results.

Altogether 271 examples of end-to-end suture are under review, and the majority of the patients have been examined at regular intervals of from four to six weeks for varying periods up to three years after the date of the performance of the suture.

As a preliminary it is convenient to consider the following general factors which may influence the progress and result.

(1) *The interval which has elapsed between the reception of the injury and the date of the suture.*—It is necessary, in connexion with this, to take into account the cause of the delay. If the operation has been deferred because of the presence of sepsis, widespread intraneural changes are likely to be encountered which will interfere seriously with, or prevent, regeneration. Excluding such a condition, a delay of twelve to eighteen months appears to have no marked effect upon the date or extent of recovery. If the interval exceeds this time, in my experience, the prognosis is not so good when the suture has been performed in the distal part of the limb; whereas, in the proximal part, a delay of two or three years does not seem to prejudice the chances of success. Suture of the median in the upper arm has been successful

in two patients where there was an interval of two and a half years. A delay of three and a half years (the longest encountered) occurred in two patients, with injury to the median at the elbow. In one the only muscle to recover voluntary power was the flexor sublimis digitorum; in the other patient it is too early yet to give an opinion, but the signs are not hopeful.

(2) *The surgical technique adopted.*—In about 80 per cent. the same procedure has been used; so that in this series this factor is almost constant, and, consequently, no opinion can be expressed upon the advantages of the various suture materials. After free exposure, the extremities have been excised until distinct nerve bundles could be seen, and then the ends brought into as perfect apposition as possible without distortion. Whenever practicable, only sheath sutures of catgut have been employed, but in the larger proportion one tension suture through the substance of the nerve was found necessary. On comparing the progress and results, with and without tension sutures, there is no doubt that the effect of the through-and-through stitch is perceptible.

For some time my surgical colleagues have used no protective material to surround the line of suture; a new bed for the nerve has been constructed, or its anatomical course modified, so that the injured segment of the nerve lies only in relation with uninjured muscles. The clinical records clearly indicate the advantages of this over any protective material, either autogenous or heterogenous. Several nerves have been re-exposed by my colleague, Mr. H. Platt, after various materials have been used, and it has been proved that neither fascia lata nor fat prevent adhesions, and in two patients, where Cargile membrane had been employed, there was a thick collar of fibrous tissue around the nerve which in one was undoubtedly causing considerable constriction.

The prognosis is bad when a satisfactory bed cannot be procured, and the nerve has to be left in relation with scar tissue, a condition unfortunately encountered frequently when the external popliteal nerve has to be sutured in the region of the head of the fibula.

Displacement of the ulnar nerve in front of the internal condyle of the humerus has not, as far as I can ascertain to date, retarded the progress.

It is somewhat surprising that the extensive freeing of the nerve necessary in this manœuvre, or in other nerves when a large defect has to be bridged, has not had a more profound effect. I have insufficient

records yet to give an opinion upon the results after the two-stage operation.

The after-treatment has been the same—constant support of the paralysed muscles, interrupted galvanic stimulation of the muscles and massage, and later faradic stimulation and muscle re-education.

(3) *The condition of the nerve found on exposure at operation.*—Those patients in whom a complete solution of continuity was discovered at operation have tended to make a more satisfactory recovery than those in whom incomplete division was found, but I am convinced that this difference is very much reduced, if not eliminated, when a liberal resection is performed. Interstitial changes are frequently very extensive after partial division, and from the histological examination of pieces of nerve excised at operation there appears to be a tendency not to resect sufficiently widely. Free resection is particularly necessary after prolonged suppuration. Contractures and other mechanical disabilities are associated commonly with incomplete division and may be responsible for imperfect functional recoveries, although good regeneration may have succeeded the suture of the nerve.

(4) *The effect of some of the more frequent complications.*—Allusion has been made to the fact that extensive and prolonged sepsis in the neighbourhood of the nerve may influence seriously the prognosis. Microscopic examination has shown that, under such a condition, intraneural changes may be discovered as distant as 8 in. above the point of injury.

Ligature of the main artery in the proximal part of the limb, as the upper part of the brachial or the axillary, tends to retard the progress and, after suture of the ulnar or median nerves, appears sometimes to reduce the extent of the recovery of function, but it is difficult to exclude the possibility of this being due to secondary changes in the muscles as a result of the ischæmia. Involvement of the main blood-vessels in the distal part of the limb, as may be anticipated, does not usually prejudice the result.

Ununited fractures of the humerus present a serious complication. In four patients exhibiting this complication suture of the musculo-spiral was performed before the ends of the bones had been fixed. Two were complete failures, the third showed merely recovery in the supinator longus and extensor carpi radialis longior, whilst the fourth progressed favourably until a bone graft operation was succeeded by a recrudescence of sepsis, which resulted in complete loss of conduction and failure of regeneration. These experiences seem to prove that

where an ununited fracture of the humerus complicates a musculo-spiral injury it is advisable to treat the bone lesion first.

II.—Analysis of Results.

(*a*) *General.*—In none of my cases of secondary suture can the result be described as perfect; in the best there has been an appreciable difference in power on comparing the two sides and at least an incomplete recovery of epicritic sensibility, even three and a half years after the date of the suture. The recovery of sensation has been particularly disappointing. More successful results have followed suture in the proximal part of the limb than the distal. Of sutures in the upper extremity, the failures in the upper arm amounted to 12 per cent., whilst in the forearm to 24 per cent. At both levels the recovery of voluntary power has been much less constant for muscles supplied by the nerve in the distal than in the proximal part of the limb. For obvious reasons muscles which have a large nerve of supply have a better chance than those in which the branch is smaller.

Exclusive of mechanical disabilities and the general factors already considered, four chief causes for imperfect recovery are readily recognized.

(1) Intraneural changes in the proximal end, which may occur irrespective of the presence of sepsis.

(2) Obstruction to the downgrowth of new nerve fibrils.

(3) Destruction of branches in addition to the injury to the nerve trunk. This is particularly prominent in wounds of the median in the upper third of the forearm.

(4) "Bad shunting" (efferent fibres passing down to afferent terminals or vice versa), which, with the most perfect surgical technique, must occur to some extent in almost all secondary sutures, since a considerable portion of the nerve trunk has to be excised and the intraneural anatomy disturbed. Such a factor will be of less consequence in the musculo-spiral than in the ulnar or median. It is possible that the persistence of Tinel's sign without any recovery of sensation may be due to afferent fibres descending efferent pathways.

(5) In addition to these there is an important but less obvious fifth cause of impaired function, which is particularly significant after suture of the median, and is probably one explanation of the poor functional result which usually follows suture of both the median and the ulnar. It is not uncommon to discover, after suture of the median, that all the muscles—when tested individually—have recovered voluntary power,

and yet the hand is of slight practical service, when the patient attempts purposive movements. The patient will often volunteer the information that when he attempts to work he finds that he loses the grip of his tools, and on inquiry it will be found that he can use the hand fairly well as long as he concentrates upon the movements, but the hand ceases to function satisfactorily as soon as he takes his eye off it. Such patients also frequently inform you that the hand is useless in the dark, or when they cannot watch what they are trying to do with it. Such complaints may be heard even when cutaneous sensibility has made a fair recovery and all the muscles, tested individually, exhibit voluntary power. It seems clear that such a disability is due to the loss of afferent stimuli from joints, muscles, tendons and other deep structures. It is only practicable to investigate directly the recovery of fibres conveying conscious impressions from such deep structures as joints, but from an investigation of the sense of posture and the appreciation of passive movements in the finger and thumb articulations supplied by the nerve, it has been found that these were generally lost, or at the best very defective even when three years had elapsed since the time of the suture. It would seem that this is very important to remember at examinations for the assessment of pensions after nerve suture in the upper limb, since the routine investigations of voluntary power and cutaneous sensibility do not, in themselves, provide sufficient information to determine the real functional capacity of the hand. It is also an awkward problem to contend with during the later stages of recovery when muscle re-education becomes of greatest importance, since it provides one of the main obstacles to the satisfactory application of this form of treatment—an obstacle which is probably insufficiently appreciated. Previous to the war our chief experience of muscle training was derived from the treatment of infantile paralysis, and consequently many who are responsible for the supervision of this form of treatment are content merely to develop an increased range of movement of the individual muscles, and fail to realize the different problem which arises in peripheral nerve injury. After injuries to the peripheral nerves there is usually a serious loss of those afferent stimuli which are of such importance for the perfect performance and adjustment of the finer and more delicate movements, and consequently in the later part of the treatment every effort must be made to develop those purposive and more complex movements which the particular patient will require when he returns to a civil occupation.

(*b*) *Explanation of charts.*—The vertical columns, 1 to 24, refer

to the months after the performance of the suture; in the last column, "delay," reference is made to any case where an interval of eighteen months or more has occurred between the date of the reception of the wound and the time of the operation, the figures express this interval in months. In all the charts reference is made, unless otherwise stated, to the return of voluntary power, since in my experience this has been more frequently discovered at an earlier date than the recovery of faradic response. The muscles are recorded by means of the initial letters, viz. :—

SL = supinator longus.

As regards sensation information is conveyed by the following abbreviations and letters :—

P = commencement of recovery of protopathic sensibility.

P.rec. = complete recovery of protopathic sensibility.

E. = commencement of recovery of epicritic sensibility.

The interrupted lines in certain columns mark the date of the last examination, unless they occur in the last column, in which case several of the patients have been under observation for further periods, but the intervals between the later examinations have been longer than four to six weeks.

(c) *Musculo-spiral nerve.*—This nerve offers the best opportunity of investigating many of the problems, since the influence of two factors, "bad shunting" and the loss of afferent impulses from joints, &c., is reduced to a minimum, and of much less significance than in such nerves as the median or ulnar, which contain a large proportion of afferent and sympathetic fibres, as well as efferent. This, together with the fact that suture of the musculo-spiral must occur in the proximal part of the limb, may account for the constancy of a good recovery.

The patients may be divided easily into three groups according to the anatomical level of the suture.

(1) In the lower third of the arm, i.e., below the level of the musculo-spiral groove, and where the trunk is lying deeply between the brachialis anticus and supinator longus. In two patients, destruction of the branch to the supinator longus was recorded at operation, and this is responsible for the absence of recovery of this muscle in patients 10 and 18. Excluding the three failures, the recovery has followed a fairly regular course, and the order of the muscles as they regain voluntary power has followed very closely the anatomical arrangement of the branches (fig. 1). In patients 12 and 19 the recovery is divided into two stages by an interval of eight months and ten months respectively, a phenomenon

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 1				S.L.	E.C.R.		E.C.U.		E.C.D.				E.O.M.P.	E.L.P.		E.B.P.								- - - -	
,, 2				S.L.		E.C.R.	E.C.U.	E.C.D.	E.O.M.P.			E.L.P.												- - - -	
,, 3					S.L.		E.C.R.			E.C.U.				E.L.P.		E.O.M.P.	E.C.D.								
,, 4						S.L.	E.C.R.	E.C.U.				E.C.D.		- - - -											
,, 5				S.L.	E.C.R.	E.C.U.	E.C.D.	- - - -																	
,, 6				S.L.	E.C.R.	^{3}E.C.D.	E.C.U.				$^{1\,2\,3\,4}$E.C.D.	E.O.M.P.	E.L.P.	E.B.P.		- - - -									
,, 7				S.L.	E.C.R.		E.C.U.	E.C.D.			E.O.M.P.		E.L.P.												
,, 8						S.L.		E.C.R.					E.C.U.		E.C.D.	E.O.M.P.			E.L.P.				E.B.P.	- - - -	
,, 9			S.L.		E.C.R.			E.C.D.			E.O.M.P.	E.L.P.													
,, 10				E.C.R.	E.C.U.	E.L.P.	E.O.M.P.	$^{1\,2}$E.C.D.																	
,, 11				S.L.	E.C.R.		E.C.U.	E.O.M.P.						E.C.D.					E.L.P.		E.B.P.			- - - -	
,, 12				S.L	E.C.R.		E.C.U.									E.C.D.		E.O.M.P.		E.B.P.				- - - -	
,, 13			S.L.	E.C.R.				E.C.D.																	
,, 14		S.L.	E.C.R.																						
,, 15				S.L.	E.C.R.			- - - -																	
,, 16			S.L.			E.C.R.	E.C.U.	E.C.D.			- - - -														
,, 17											E.C.R.					E.O.M.P.	E.L.P.			- - - -					
,, 18			E.C.R.		E.C.U.																				
,, 19								E.C.R.	E.C.U.											E.C.D.		E.O.M.P.		E.L.P.	
,, 20			E.C.R.																						
,, 21			E.O.M.P.																						
,, 22				S.L.																					
,, 23													Failure							- - - -					
,, 24												Failure							- - - -						21
,, 25														Failure								- - - -			19

Chart I.—Musculo-spiral nerve (lower third of arm).

The numerals above E.C.D. refer to the first (index), second, third and fourth (little) fingers.

which will be found in several of the other charts. A possible explanation is that the young fibrils, which subsequently supply the muscles late to recover, have encountered some obstruction, and the delay is due to the time spent in penetrating this.

Of the three failures, one had previously had a nerve graft inserted to bridge a large defect, which procedure had proved unsuccessful; in the second, prolonged suppuration had prevented surgical interference until twenty-one months after the injury; whilst in the third there is clinical evidence of a neuroma at the site of the suture, indicating that some regeneration has occurred, but for some reason, as yet undetermined in this case, the young fibrils have been prevented from penetrating the distal end.

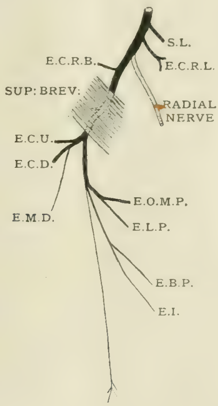

FIG. 1.

(2) The sutures performed in the middle third of the arm, in the musculo-spiral groove, include the four complicated by an ununited fracture of the humerus to which reference has previously been made.

Patient 46 was seen for the first time eighteen months after operation, and there was then no recovery of voluntary power, but a faradic response was present in the supinator longus and the extensors of the wrist and fingers. With suitable treatment, he is recovering from this functional disability.

With the exception of those patients in which an ununited fracture of the humerus occurred, there was only one failure in this series,

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 26				S.L.	E.C.R. E.C.U.				E.C.D.	E.O.M.P.	E.L.P.			E.B.P.			----								
,, 27				S.L.	E.C.R.	E.C.U.					E.C.D.		E.O.M.P.												
,, 28					S.L.		E.C.R. E.C.U.			E.O.M.P.		E.C.D.				E.L.P.								E.B.P.	
,, 29					S.L.	E.C.R.		E.C.U.	E.O.M.P.																
,, 30						S.L.		E.C.R.			E.C.U.			----											
,, 31									E.C.R.		S.L.				E.O.M.P.						----				
,, 32					S.L.	E.C.R.		E.C.D.	E.C.U.		E.O.M.P.	E.L.P.		E.B.P.			----								
,, 33					E.C.R.	S.L.	E.C.U.				----														
,, 34					S.L.	E.C.R.		E.C.U.																	
,, 35				S.L.	E.C.R.		E.C.U.		----																
,, 36			S.L.	E.C.R.		----																			
,, 37					S.L.	E.C.R.																			
,, 38				S.L.		E.C.R.																			
,, 39			S.L.			E.C.R.																			
,, 40													E.C.R.		E.C.U.	3 4 E.C.D.							----		
,, 41								S.L.	E.C.R.		E.C.U.				E.O.M.P.		E.C.D.		E.L.P.					----	
,, 42				S.L.																					
,, 43																					----	All	acting		
,, 44					E.C.R.		----																		
,, 45																				S.L.	2½ years E.C.R.	E.C.U.	E.C.D.	E.O.M.P.	
,, 46												Faradism S.L.	E.C.R.	E.C.U.	E.C.D.			Nil Vol:			----				
,, 47						S.L.	E.C.R.					Bone graft went septic					—	Failure					----		
,, 48												E.C.R.	S.L.						E.C.U.		Ununited Fracture				
,, 49																	Failure				Ununited Fracture				
,, 50														----		Failure		—	Ununited			Fracture			
,, 51																		Failure			(2 yrs.)		Bulb.	----	24

Chart II.—Musculo-spiral nerve (middle third of arm).

and in this patient an interval of two years had elapsed before the suture was performed.

(3) The rapidity and completeness of recovery in the first four patients on Chart III is of importance and interest, since the suture in each was above the level of the groove, and may be considered as being in the axilla.

Patient 56 collapsed during the operation, and the ends of the nerve consequently were merely anchored by one through-and-through stitch, yet voluntary power has slowly returned in the supinator longus and extensors of the wrist.

Chart IV demonstrates the date of recovery of voluntary power in the various muscles at the three levels, and reference to it shows that recovery is proportionately more rapid the nearer the suture is to the spinal cord. In the lower third of the arm the supinator longus recovered most frequently about the fourth month, whilst in the upper third about the seventh month, yet the regenerating fibrils in the latter had to grow more than three times the distance traversed in the former. Similar results are to be seen in the other muscles. Are we to conclude from this that a nerve fibre regenerates more rapidly and readily the nearer the section has been made to its anterior cornual cell? The better prognosis for sutures in the upper arm compared with those in the forearm supports such a contention.

From the foregoing results it may be concluded that a good functional result is to be anticipated after secondary suture of the musculo-spiral nerve, provided there is no special complication or contra-indication.

The extensors of the wrist usually re-

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
Patient 52					S.L.	E.C.R.	E.C.U.	E.C.D.			E.O.M.P.	E.L.P.			E.B.P.									
,, 53							S.L.	E.C.R.	E.C.U.									E.O.M.P.	E.L.P.	E.C.D.				
,, 54							S.L.		E.C.R.		E.C.U.	E.O.M.P.					E.C.D.			-----				
,, 55							S.L. E.C.R.		E.C.U.	E.O.M.P. E.B.P.		E.C.D.		E.L.P.							-----			
,, 56			Triceps							S.L.			E.C.R.			E.C.U.	-----							
,, 57						S.L.	E.C.R.	-----																
,, 58						E.C.R.	-----																	

CHART III.—Musculo-spiral nerve (upper third of arm).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
S.L.		1.	3.	9.	1.	2.																		
E.C.R.			3.	2.	8.	2.	2.	2.			1.													
E.C.U.					2.	1.	7.	1.	1.	1.			1.											
E.C.D.						1.	1.	6.	1.			1.		1.	1.	1.	1.			1.				
E.O.M.P.			1.				1.	1.	1.		2.	1.	1.			3.		1.				1.		
E.L.P.						1.						2.	2.	2.			1.		2.	1.				1.
E.B.P.														1.		1.					1.		1.	

Lower third (twenty-five cases).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
S.L.			2.	5.	5.	3.		1.			1.		1.											
E.C.R.				1.	5.	6.	2.	1.	2.			1.	1.											
E.C.U.					1.	1.	3.	2.	1.		2.				1.				1.					
E.C.D.								1.	1.		1.	1.				1.	1.							
E.O.M.P.									1.	2.	1.		1.		2.									
E.L.P.											1.	1.				1.			1.					
E.B.P.														2.										1.

Middle third (twenty-six cases).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
S.L.					1.	1.	3.			1.														
E.C.R.						2.	2.	1.	1.				1.											
E.C.U.							1.		2.		1.					1.								
E.C.D.								1.				1.				1.				1.				
E.O.M.P.										1.	1.	1.						1.						
E.L.P.												1.		1.					1.					
E.B.P.											1.				1.									

Upper third (seven cases).

CHART IV.—To show date of recovery of voluntary power of the various muscles at the three levels.

gain full power, and the extensor communis digitorum recovers satisfactorily, although the extension of the index is not infrequently imperfect, and it is almost the rule to find that the patient is unable to extend this finger alone at the metacarpo-phalangeal joint.

The extensors of the thumb rarely show such a perfect recovery, the extensors brevis pollicis being most commonly paretic, which is the common cause for the imperfect extension of the thumb and the difficulty experienced in performing such a movement as opening a pair of scissors.

The extensors of the wrist have never been found to recover their synergic action before that of a prime mover; in fact, in end results it has been almost constantly found that the grasp was weak, owing to the loss of the synergic extension of the wrist, unless this movement had been carefully developed during the period of muscle-training. A number of patients have returned to hospital complaining of weakness of the hand grasp, although the nerve had regenerated well and the extensors of the wrist were quite powerful, and this has been found to be due simply to the loss of their synergic action. It is hardly to be expected that there would be a spontaneous recovery of the synergic action unless the same set of fibres innervated the extensors of the wrist after the suture as before, a condition which is not likely to occur frequently.

(*d*) *Median nerve.*—Unfortunately many patients, in whom this nerve had been sutured at the wrist, have been lost, as they were able to procure light employment, and it was not often possible to keep them under observation for long periods. In quite a large proportion only an incomplete recovery of protopathic sensibility has occurred, and voluntary power in the abductor brevis pollicis has only been found in some 40 per cent. of those in which sufficient time has elapsed to expect this to be possible.

Only the abductor brevis pollicis of the thenar group has been mentioned, because the flexor brevis pollicis is composed of several slips, some supplied by the median and others by the ulnar, which are subject to considerable individual variation; and it is consequently difficult to give a reliable opinion as to the presence of voluntary power in the flexor brevis pollicis from a clinical examination.

Of the sutures in the forearm (Chart VI) in patients 82 to 90, this was performed below the level of the anterior interosseous branch; in two (patients 91 and 92) above this branch, but distal to the supply to the flexor carpi radialis and pronator radii teres; whilst in the

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 59				P.							P.rec.							A.B.P.						-----	
,, 60					P.									P.rec.	A.B.P.	E.		-----							
,, 61					P.							P.rec.			E.			A.B.P.						-----	
,, 62					P.			P.rec.		-----															
,, 63					P.		A.B.P.				-----														
,, 64								P.	A.B.P.		P.rec.					E.				-----					
,, 65			P.					-----																	
,, 66				P.					-----																
,, 67				P.				P.rec.			-----														
,, 68				A.B.P.	P.							-----													
,, 69				P.											-----										
,, 70				P.		A.B.P.				P.rec.		-----													
,, 71			P.	A.B.P.						-----															
,, 72				P.						-----															30
,, 73				P.								-----													
,, 74			P.			-----																			
,, 75			P.			-----																			
,, 76			P.			-----																			
,, 77					P.												-----								
,, 78				P.								P.rec.													
,, 79					P.								-----												19
,, 80																		Failure							
,, 81																		Failure							18

CHART V.—Median nerve (wrist).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 82			^{1}F.S.D.	P.			-----																		
,, 83				^{1}F.S.D.	P.							A.B.P.													
,, 84				A.B.P.	^{1}F.S.D.				P.				E.				-----								
,, 85					^{1}F.S.D.						P.	A.B.P.												-----	
,, 86				P.		^{1}F.S.D.		-----																	
,, 87			P.	^{1}F.S.D.					-----																
,, 88						P.		^{1}F.S.D.																	20
,, 89							^{1}F.S.D.		P.					-----											
,, 90		P.			^{1}F.S.D.																				
,, 91				F.S.D.	P.			F.P.D.		F.L.P.		E.		-----											
,, 92							P.	F.L.P.		F.S.D.	F.P.D.	Prec.			A.P.B.			E.						-----	
,, 93				F.S.D.	P.R.T.			F.L.P.	P.						Prec.										
,, 94				F.C.R.	$^{2.3.4.}$F.S.D.	P.			F.L.P.	F.P.D.								^{1}F.S.D.						-----	
,, 95				F.L.P.														F.S.D.						-----	
,, 96			P.	F.S.D.														-----							41
,, 97										P.			F.C.R. F.L.P.				P.R.T.			-----					
,, 98			P.R.T.	F.S.D. P.	F.C.R.	F.P.D. F.L.P.																			
,, 99					P.R.T.	F.C.R.					P.	-----													
,, 100						F.S.D.					F.L.P.		-----												20
,, 101										P.		-----													
,, 102				P.		-----																			42
,, 103																	All	acting		vol: –	3½ years Anaesth: -		only index		
,, 104																	All	acting (except		F.S.D.) -	2½ years	Anaesth: distally			
,, 105																	All	muscles	acting	-	2½ years Anaesth:		2 fingers		
,, 106															Nil	vol:*	F.C.R. -	F.S.D. -	F.P.D. -		resp: to Farad:				
,, 107																		Failure							

CHART VI.—Median nerve (forearm).

Patients 82 to 90 below anterior interosseous branch; patients 91 and 92 above anterior interosseous, but below branches to flexor carpi radialis and pronator radii teres; patients 93 to 107 above all muscular branches. Numerals above flexors of fingers refer to first (index), second, third and fourth (little) fingers.

* Date when first examined after suture.

remainder it was done proximal to all the muscular branches. In several of the latter group, branches, in addition to the nerve trunk, were injured, and this was responsible for the failure of recovery of some of the muscles.

Patients 103, 104, and 105 are later results, and were first examined some considerable time after the suture.

Patient 106 is another example of a hysterical loss of power; three of the muscles responded to faradism, and there was a sensory loss of the "glove" type.

The results in Chart VII are comparable to the satisfactory recoveries already seen after suture of the musculo-spiral in the proximal part of the limb as regards completeness, regularity, and rapidity of recovery.

The late commencement of improvement and the slower progress in patient 116 is probably due to the ligature of the axillary artery, since no other reason for this can be determined.

(*e*) *Ulnar nerve.*—The disappointing results which follow suture of this nerve are now well known, but it is still difficult fully to understand the reason for the poor prognosis. The risk of "bad-shunting" is clearly considerable, and may be the chief explanation; and from this it is easy to understand how the intrinsic muscles of the hand, with their small nerves of supply, run a greater risk of not receiving any efferent fibres than the larger muscles. At one time it was imagined that a poor blood supply might be the cause of the incomplete recovery or failure; but, from dissections made after special injection of the blood-vessels of the limb, I find that the ulnar nerve has quite as efficient, and often a richer, blood supply than the median.

Many of the patients, in which the suture was performed in the forearm, have been lost owing to the reason mentioned in the case of suture of the median at the wrist.

In three out of the seven failures in Chart VIII it is of interest to notice that there was a delay of more than eighteen months before the operation was done.

After suture in both the arm and the forearm the recovery of voluntary power in the small intrinsic muscles of the hand was particularly poor and disappointing.

Brouwer [1], in a recent paper, advances a hypothesis which may prove to be a true explanation of the bad prognosis for the intrinsic muscles after lesions of the median and ulnar nerves. He found, in neuritis of the median, that in a large number of patients there was a

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 108						P.R.T.	F.C.R.				P.	F.L.P.		F.S.D.				F.P.D.			A.B.P.	Prec.		----	
,, 109					P.R.T. F.C.R.	P.		2.3.4. F.S.D.						1. F.S.D.	Prec.					----					
,, 110					P.R.T.	F.S.D. P.	F.C.R.							F.L.P.	Prec.	A.B.P.					F.P.D.			----	
,, 111				P.	P.L.	F.C.R.	F.S.D.	F.P.D.		----															
,, 112				P.		2.3.4. F.S.D.	1. F.S.D.								F.C.R.		----								
,, 113					P.	P.R.T.	F.C.R.	2.3.4. F.S.D.					----												
,, 114					P.R.T. F.C.R.	F.S.D.	F.L.P. P.	F.P.D.																	
,, 115						P.R.T. F.C.R.					P.	F.L.P. F.S.D.						F.P.D.						----	
,, 116							P.R.T.				F.S.D.	F.L.P.	F.C.R.												
,, 117					P.R.T.	F.S.D.		F.L.P.										F.P.D.		A.B.P.				----	
,, 118				P.		P.R.T.	F.C.R.																		
,, 119			P.R.T.	F.S.D. P.	F.C.R.	F.P.D. F.L.P.																			
,, 120			P.R.T.		P.		F.C.R.	F.S.D.	A.B.P.								Prec.								
,, 121							P.R.T.	F.C.R.	2.3.4. F.S.D.			----													
,, 122				P.		P.R.T.	F.C.R.	F.S.D.		Prec.															
,, 123			P.R.T.	P.		2.3.4. F.S.D.		1. F.S.D.			F.L.P.														31
,, 124					P.																				
,, 125																			P.R.T.	- P.L.	F.C.R.-	F.S.D.-	* F.L.P.-	Prec.	
,, 126																P.R.T.	- F.C.R.	- F.S.D.		- F.P.D. *	- Prec.	- E.		----	19
,, 127														P.R.T.	- P.L.	- F.C.R.	- F.S.D.	* - F.L.P.		- A.B.P.	- Prec.				
,, 128												P.R.T.-	F.C.R.	* F.S.D.-	F.L.P.										
,, 129			P.R.T.	F.C.R.						F.S.D.	F.L.P.		----												
,, 130							F.C.R.	----																	

Chart VII.—Median nerve (arm).

* Date when first examined after suture.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 131							P.							A.M.D.		- - - -									
,, 132						P.					P.rec.		A.M.D.									E.		- - - -	
,, 133						P.				A.M.D.		E.			- - - -										
,, 134			P.			A.M.D.		P.rec.	Inter.																
,, 135							P.						- - - -												
,, 136							P.	A.M.D.																	
,, 137			P.			A.M.D.	P.rec.			Inter.										- - - -					
,, 138															A.M.D.	P.	Inter.								
,, 139								P.																	
,, 140						P.												P.rec.			E.	- - - -			
,, 141					P.		- - - -																		
,, 142					P.	A.M.D.																			
,, 143			P.				- - - -																		
,, 144						P.			- - - -																
,, 145					P.	- - - -																			
,, 146				P.				- - - -																	
,, 147				P.				- - - -																	21
,, 148					P.																				
,, 149								P.						- - - -											
,, 150					P.					- - - -															22
,, 151				P.		- - - -																			18
,, 152						P.						P.rec.			- - - -										
,, 153																				* A.M.D.	only.				
,, 154																				A.M.D.	- Inteross:		3 years No Sens: rec.		
9 No evidence of regeneration 6 - 10 mths.																									
7 Failures																									

CHART VIII.—Ulnar nerve (forearm).

Distal to branches to flexor carpi ulnaris and flexor profundus digitorum.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 171							P.		A.M.D.											P.rec.				-----	
„ 172			F.C.U.	F.P.D.	P.		A.M.D.		-----																
„ 173			F.C.U.	F.P.D.		P.								-----											
„ 174				P.						----															
„ 175				F.C.U.			F.P.D.				P.													-----	
„ 176						F.C.U.																			
„ 177						F.C.U.														P.			A.M.D.		
„ 178				F.C.U.									F.P.D.				A.M.D.							-----	
„ 179						F.C.U.																			
„ 180			F.C.U.						F.P.D.							-----									
„ 181			F.C.U.			-----																			
„ 182								P.																	
„ 183		F.C.U.			F.P.D.			A.M.D.																	
„ 184											F.C.U.	P.	F.P.D.									P.rec.			
„ 185				F.C.U.	P.	F.P.D.																			
„ 186			F.C.U.		F.P.D.			A.M.D.		-----															
„ 187						P.					-----														18
„ 188									F.C.U.																
„ 189				F.C.U.	F.P.D.																				
„ 190				F.C.U.	P. F.P.D.					A.M.D.		-----													
„ 191				F.P.D.								-----													
„ 192				F.P.D.	P.																				18
„ 193		F.C.U.	F.P.D.		P.								-----					-----							
„ 194			F.C.U.	F.P.D.		----																			
„ 195						F.C.U.		F.P.D.					----												
„ 196			F.C.U.										----												
„ 197				F.C.U.			----																		
„ 198			F.P.D.	P.																					
„ 199				F.C.U.				----																	
„ 200																						F.C.U. only *			
„ 201																			F.C.U. * & F.P.D. only						
„ 202																	F.C.U. & * F.P.D. only								
„ 203																		F. C. * U. only							
„ 204																		F. C. U.-F. * P. D. & Inteross: P.rec.							18
„ 205																				F.C.U. - F.P.D. - A.M.D. & P. *					
„ 206																			F.C.U. & F.P.D. only				3½ years		
								8 Failures & 2 nil at 6 months																	

CHART IX.—Ulnar nerve (arm).

loss of voluntary power and faradic response in the thenar muscles—although the other muscles supplied by the median were perfectly normal, and ascribed this greater vulnerability of the muscles of the thumb, or their nerve branches, to the "finer construction of the thumb and higher functional significance which it has received in the phylogenesis." Whether Brouwer's suggestion is the sole or part explanation, or whether the infrequent recovery of the intrinsic muscles is due to their distance from the cells of the anterior cornu, future experimental and clinical research must finally decide, but reference to the charts and consideration of the recovery of afferent, as well as efferent, fibres suggests that there is evidence that the latter is, at any rate, a factor.

The complete separation of the extensor brevis pollicis from the extensor ossis metacarpi pollicis is a human characteristic, and since the extensor brevis pollicis was found to recover less frequently than either of the other extensor muscles of the thumb after suture of the musculo-spiral, it would appear, at first sight, that this was further support of Brouwer's contention that more recent acquisitions are more vulnerable than ancient ones. But this support cannot be accepted without qualification, because the extensor brevis pollicis is innervated at a much lower level than the other two muscles, and its nerve of supply is considerably smaller than either that to the extensor ossis metacarpi pollicis or the extensor longus pollicis (fig. 1); and, furthermore, it is significant that the extensor brevis pollicis recovered in 16 per cent. of all the sutures in the lower third of the arm, 17 per cent. in the middle third, and in 29 per cent. in the upper third, which figures strongly suggest that the distance of the suture from the spinal cord exerts an important influence upon the prognosis.

(*f*) *Sciatic nerve.*—In this group the one instructive point to direct attention to is that in one patient only, as yet, has there been recovery of the tibialis posticus and the flexors of the toes, although the gastrocnemius, which has a much larger nerve supply, almost invariably recovers, even more readily than any of the muscles supplied by the external popliteal segment.

Patient 247 is an example of the two-stage operation, and after an interval of eleven months there is no clinical evidence of any regeneration.

(*g*) *External popliteal nerve.*—Five failures out of twenty-one is rather a surprising proportion for the external popliteal, as the prognosis after suture of this nerve has often been considered almost as favourable

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 217											Gastr:			E.L.D.	Peron:	T.A.		-----							
„ 218								P.	Gastr:	T.A.		-----													
„ 219				P.										Gastr:											
„ 220													Gastr:		T.A.		E.L.D.			Peron:				-----	
„ 221												T.A.								Peron:	Gastr:		-----		
„ 222																P.		Gastr:						-----	
„ 223									Gastr:	T.A.												All	acting	2½ yrs.	
„ 224														Gastr:		-----									
„ 225					Gastr:												P.						27 mths. Same		
„ 226						Gastr:	T.A.					E.L.D.			P.										
„ 227					Gastr:	P.				T.A.							E.L.D.	-----							
„ 228							P.	Gastr:	T.A.	E.L.D.		-----													
„ 229					P.							Gastr:	T.A.	-----											
„ 230										Gastr:											P.			-----	
„ 231					P.			Gastr:	T.A.		----														
„ 232				P.		T.A. Gastr:	E.L.H.																		
„ 233								Gastr:			----														
„ 234					Gastr:	P.					Prec.														
„ 235					P.										Gastr:			T.A.			-----				
„ 236						P.		Gastr:																	
„ 237					Gastr:			T.A.	-----																
„ 238								Gastr:					----												
„ 239													----												
„ 240				P.		----																			
„ 241				P.	Gastr:						----														24
„ 242				P.			-----																		30
„ 243																						Gastr:	2½ years only		
„ 244																				Failure					
„ 245																						Failure			
„ 246																						Failure			
„ 247											----														

CHART X – Sciatic nerve.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	Delay
Patient 248						P.		T.A.				E.L.D.	Peron:													Prec.	
,, 249											P.		T.A.		Peron:						----						
,, 250					T.A.										P.	Peron:	E.L.D.	E.L.H.								----	
,, 251							T.A.	E.L.D.																----			
,, 252				P.		T.A.	Peron:					E.L.D.								Prec.						E.L.H.	
,, 253						P.							----														
,, 254				P.		T.A.							Prec.	----													
,, 255						T.A.	Peron:																				
,, 256										P.					Peron:		Prec.								T.A.	----	
,, 257			P.		T.A.	E.L.D.	Peron:		Prec.					E.L.H.				E.						----			
,, 258							----																				
,, 259							----																				
,, 260													----														27
,, 261																					Nil vol:		All respond to Farad:		*		
,, 262										----																	
,, 263						T.A.		P.		Peron:			E.L.D.		----												
,, 264				Peron:										----													
,, 265																				Failure							
,, 266																							Failure		2 years		
,, 267																								Failure	3 years		
,, 268																					Failure		2 years				
,, 269																							Failure		2 yrs.		

CHART XI.—External popliteal nerve.

as that of the musculo-spiral. An explanation of this, however, is forthcoming when we are reminded of the difficulty of procuring a good bed for the nerve in the region of the head of the fibula, and consequently it is suggested that secondary adhesions and involvement of the nerve in scar tissue are responsible for such a very high proportion of failures.

Patient 261 is a third example of a functional loss of power, although there is good evidence of innervation of all the muscles supplied by the external popliteal.

A comparison of the very rapid and complete recovery of patient 257, with that of the others, is of considerable interest, because his may be considered a primary suture, since only a very slight laceration was found at operation. The recovery, two years after suture, was practically perfect.

(*h*) *Internal popliteal nerve.*—In both patients the functional result has been satisfactory, but in neither has there been any recovery of sensation.

III.—Conclusions.

(I) The following appear to be the most important factors which may influence the prognosis:—

(1) Sepsis.

(2) Site of the suture, the prognosis being more favourable the nearer the suture is to the spinal cord.

(3) A delay of more than eighteen months before the suture is performed in the distal part of a limb prejudices the chances of recovery, but is not of so much consequence in the proximal part.

(4) "Bad-shunting" is probably a common cause of imperfect recovery in such nerves as the ulnar.

(5) Destruction of branches of the nerve, in addition to injury to the trunk, may be a cause of incomplete recovery.

(6) Ununited fractures, especially of the humerus in injuries to the musculo-spiral, are a serious complication. Evidence appears to be in favour of primary fixation of the bone.

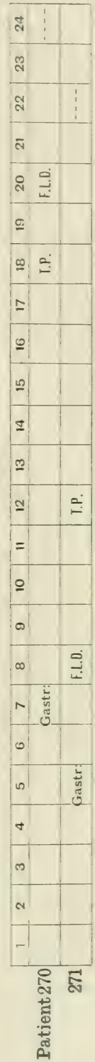

Chart XII.—Internal popliteal nerve.

(7) Ligature of the main artery in the proximal part of a limb may delay and limit recovery.

(8) Imperfect recovery of afferent fibres from such structures as joints, muscles and tendons, is probably an important cause of limited functional recovery of the hand for the performance of purposive actions, although the voluntary power of individual muscles may be good.

(II) The intrinsic muscles of the hand and the muscles in the distal part of a limb recover less frequently than the larger muscles in the proximal part of a limb. Probably several factors contribute to the cause of this :—

(1) Distance which the young nerve fibrils have to travel away from their trophic centre in the spinal cord to reach the muscle.

(2) The size of the nerve of supply. The larger the nerve the greater the chance the young fibrils must have of reaching the muscle.

(3) Possibly the phylogenetic history of the muscles, since there does seem to be some support of the hypothesis that more recent acquisitions are the more vulnerable.

(III) The following points bearing on the surgical technique can be deduced :—

(1) It is advisable to avoid the through and through suture, whenever practicable, as proved also by the experimental work of Sargent and Greenfield [2].

(2) The construction of a new bed for the nerve, after suture, is preferable to any autogenous or heterogenous protective material. The unsatisfactory bed after suture of the external popliteal nerve, in the vicinity of the head of the fibula, is probably a cause of many failures.

(3) Displacement of the ulnar nerve in front of the internal condyle of the humerus or widespread freeing to procure apposition of the ends does not appear to retard the recovery.

(4) Liberal resection of the injured ends is essential and particularly necessary if sepsis is prominent.

(IV) No recovery after the performance of secondary suture could be regarded as perfect. The chief causes of the imperfect result after *secondary* suture appear to be :—

(1) The fact that an appreciable portion of the nerve trunk has to be resected causes inevitably a disturbance of the intraneural anatomy, however carefully the surgical procedure is carried out.

(2) After secondary suture the nerve is making a *second* effort to regenerate, the first attempt being represented by a neuroma or outgrowths along fascial planes.

(3) The almost constant occurrence of one or more of the foregoing factors which have been found to influence the prognosis.

REFERENCES.

[1] Brouwer, B. "The Significance of Phylogenetic and Ontogenetic Studies for the Neuropathologist." *Journ. of Nerve and Mental Dis.*, February, 1920, vol. li, p. 113.

[2] Sargent, P., and Greenfield, J. G. "An Experimental Investigation of Certain Materials used for Nerve Suture," *Brit. Med. Journ.*, September, 1919, vol. ii, p. 407.

MICROGYRIA AND ITS EFFECTS ON OTHER PARTS OF THE CENTRAL NERVOUS SYSTEM.

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MICROGYRIA, which has hitherto been usually known under the name of hemiatrophy of the brain or arrested development of the nervous system, has been comparatively rarely described, and merits further study.

The following case came ultimately into the hands of Dr. Mott, to whom I would here cordially express my thanks for kindly allowing me to investigate the material and for many valuable suggestions during the course of the work.

SUMMARY OF THE CASE.

Vascular lesion of the right cerebral hemisphere involving chiefly the right centro-parietal region of the cortex with portions of the frontal and temporal lobes and portions of the right basal ganglia. Atrophy and arrested development of the right pyramidal tract, right mesial filet and associated structures in the mid-brain, pons, medulla and spinal cord. Atrophy of the opposite (left) side of the cerebellum with some of its various nuclei and peduncles. Atrophy of the left side of the spinal white and grey matter. Diminution in number of motor cells chiefly in the cervical and lumbar enlargements.

CLINICAL HISTORY.

Since earliest life the patient had suffered from left hemiplegia and mental hebetude. The mental deficiency steadily increased and became early associated with unilateral and epileptiform convulsions of the ordinary type. Sight and hearing were normal but the above symptoms progressed and terminated fatally in his thirty-sixth year.

Brain.—In the brain the coarse deviations from normal are best seen by a reference to the accompanying photographs. There is a remarkable difference in the size of the two hemispheres, the right being about two-thirds as large

Fig. 1.

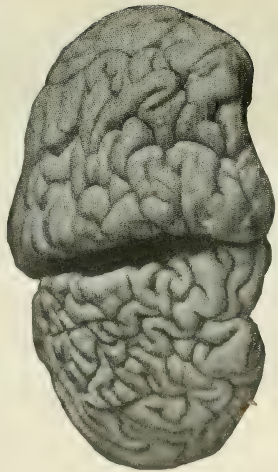

Fig. 2.

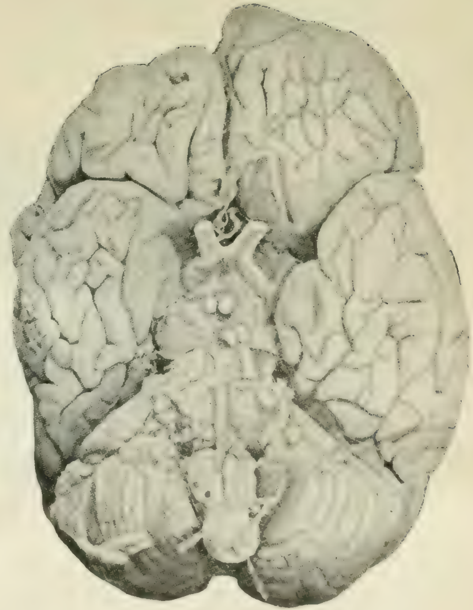

Fig. 3.

as the left, which is normal. The stress of the disease has fallen chiefly on the centro-parietal region of the right side, less on the frontal and temporal and least on the occipital lobe. The convolutions in the affected areas are shrunken in all three dimensions, i.e., they are shorter, narrower and less deep than normal and wanting both in grey and white matter. The corpus callosum is thinner than in health, but the olfactory lobes, optic tracts and cranial nerves are unaffected.

The mode of investigation adopted was to remove pieces of certain selected areas of the cerebral cortex, cerebellum and spinal cord and to stain them by various methods for minute microscopic examination of the cells and fibres. The remainder of the brain and portions of the spinal cord were cut in serial section and stained by the Weigert-Pal process, certain of these sections being again counterstained by alum, cochineal or other suitable colouring matter.

Microscopic examination of the cerebral hemisphere showed that everywhere in the affected region the cortex is thinner than normal and this most of all in the right ascending frontal convolution. All the cortical layers are wasted, but especially the pyramidal and fourth layer.[1] In the leg and body areas the very large, so-called Betz cells are not only much diminished in number and in size but have fewer processes than in health and all of them present some degenerative change. In several sections of this region, only one group of moderately good Betz cells could be found. These were situated on the flat external surface of the right ascending frontal convolution (not in the depth of the Rolandic fissure), and even they were stunted and had fewer processes than natural and showed other signs of degeneration. The right arm area presented much the same appearance as above but the pyramidal layer was not quite so much affected, In the corresponding regions of the left cerebral cortex the histological appearances were normal. The pyramidal and Betz cells were abundant and of healthy structure and no signs of hypertrophy or compensatory overgrowth was traceable. In the right facial area all layers are thin and there are very few large cells in layer 4, whereas the left facial area seems normal. In the right ascending parietal convolution all layers are much wasted except the granular layer, which looks healthy. The right first frontal gyrus has less than half its normal depth and the pyramidal layer is especially poor, whereas the left first frontal is normal. The temporal convolutions are histologically fairly normal, but the pyramidal layer is distinctly better seen in those of the left side than of the right. The visuo-sensory area in the calcarine region of the occipital lobe

[1] The classification of the layers of the cerebral cortex here adopted is that given by Mott [1], at a meeting of the Ophthalmological Society of London, Nov., 1904.

presents no definite abnormal change on either side but in the right visuo-psychic area the pyramidal layer is distinctly poorly developed.

With regard to the changes in the nerve fibres of the cerebrum, it is noteworthy that the incidence of the disease is chiefly on the efferent

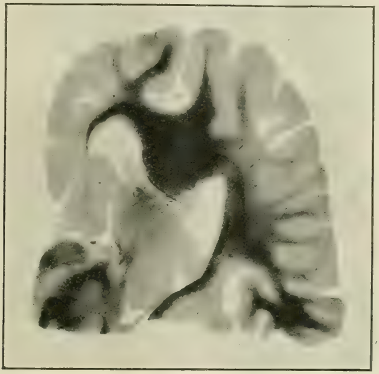

FIG. 4.

FIG. 5.

projection fibres of the affected side, which are much diminished in number, and present signs of wasting and sclerosis. The tangential fibres of the cortex and the fine network in the inner line of Baillarger

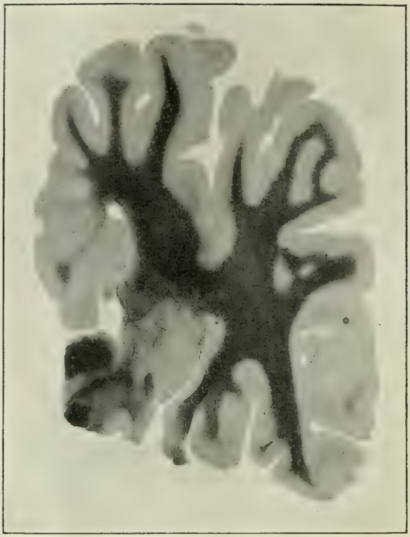

Fig. 6.

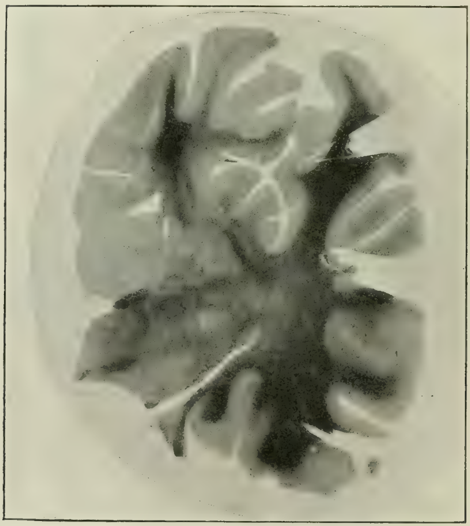

Fig. 7.

are also slightly though definitely affected in the same way. On the other hand, the fibres forming the associational bundles are not at all, or, if at all, scarcely to an appreciable extent.

In the opposite (left) cerebral hemisphere, as already remarked, no compensatory increase of cells could be found, and the nerve fibres seemed in every way normal in number and appearance. The corona radiata on the right side is much smaller than on the left (roughly about half). The right internal capsule is not only much reduced in size in its fronto-caudal plane, but also laterally where its posterior limb (the limb chiefly affected) measures from $2\frac{1}{2}$ to 3 mm., as compared with 5 to $5\frac{1}{2}$ mm. for the healthy side. In its vertical dimensions the right internal capsule is only very slightly reduced. It contains few medullated nerve fibres, and shows all the ordinary signs of atrophy and sclerosis.

The corpus striatum presents only very slight deviation from normal. The nucleus caudatus seems healthy throughout, but the lenticular nucleus, and especially the globus pallidus, are slightly wasted and not quite so rich in fibres as on the normal (left) side. On the affected side the optic thalamus is smaller both antero-posteriorly (i.e., fronto-caudally) and laterally than should be the case, and owing to a marked diminution in the number of its nerve fibres its nuclei are all less distinct than normal. The paucity of fibres is especially striking in the lateral nucleus of the right as compared with that of the left optic thalamus. Moreover, though the right arcuate nucleus is fairly well delineated the "centre-median" of Luys is scarcely traceable, and the red nucleus is less than half the size of that of the right side, and extremely poorly provided with fibres. On the left side the optic thalamus is healthy, and all its nuclei are well seen, notably the dorsal, lateral and median nuclei, the centre of Luys, nucleus arcuatus, and red nucleus and corpus subthalamicum, all of which are well seen and differentiated.

The island of Reil on the right side is smaller than normal, and its convolutions imperfectly developed. The claustrum, together with its capsula externa on its inner side, and its capsula extrema on its outer side, are well marked but slightly thinner than on the normal side. Moreover, the two capsules diminish in thickness from above down (i.e., dorso-ventrally), so that in their lower halves they are scarcely represented, and their junction below at the base of the island of Reil with the inferior longitudinal bundle and the medullary fibres around the anterior commissure is not traceable.

In the right temporal lobe the medullary fibres are generally less numerous than on the normal side, though the auditory radiations

extending as a large bundle of well stained fibres from the lateral margin of the middle geniculate body to the first temporal convolution are well marked. Similarly the general wealth of fibres in the occipital lobe is less than on the normal side, yet the optic radiations extending from the lateral geniculate body around the posterior descending horn of the lateral ventricle to the visuo-sensory area of the calcarine region are also well marked. Moreover, standing out in marked contrast to the remainder of the fibres of the temporo-occipital lobe, is the inferior longitudinal bundle which has abundant well stained fibres forming in these transverse (frontal) sections a well defined sickle-shaped mass which has its broader end above at the base of the island of Reil, and curves externally around the descending horn of the lateral ventricle, tapering away to a fine point of fibres in the region of the nucleus amygdala.

It will be seen on reference to the accompanying photographs that the third ventricle and the lateral ventricles, with their cella media, are moderately distended. This porencephalic condition still further reduces the space, which in the left hemisphere is occupied by healthy brain tissue.

Midbrain.—On the right side there is marked atrophy of the pyramidal, and less so of the middle fillet fibres. The red nucleus, as previously mentioned, is smaller than normal, and the perpendicular fibres of the crus, especially those lateral to the corpus subthalamicum, are distinctly less numerous than in the healthy hemisphere. The lateral fillet, the middle and lateral geniculate body, the anterior and posterior corpora quadrigemina on the affected side present no deviations from normal.

Pons.—The tegmentum on the right side is smaller than on the left and the middle fillet fibres are here contrasting with the middle fillet in the medulla much wasted and form a band about half as deep and not quite so broad as that of the opposite side. Also the longitudinal fibres in the mesial portion of the middle fillet are notably very much diminished in numbers, so that the nucleus reticularis tegmenti seems not only to occupy the position which should be chiefly filled by them, but actually to extend laterally, forming a thin band of grey matter with middle fillet fibres dorsally and ventrally to it. The central tegmental tract and the posterior longitudinal bundle seem slightly smaller on this side than on the left, whereas the fibræ paramedianæ are slightly less marked and the superior cerebellar peduncle distinctly less marked on the left side than on the right. The lateral fillets and

lateral pontine bundles are equal on the two sides, and with the above exceptions all the remaining structures and longitudinal systems seem normal in the tegmentum of both sides.

In striking contrast with the wasted (or lesser developed) tegmentum of the affected side is the larger development of the crural portion on that side. This is obviously due to the fact that in contra-distinction to the cerebral hemispheres the right half of the cerebellum is healthy whilst its left half is wasted. For, as is well known on physiological and anatomical grounds, one half of the cerebrum is in closest relationship functionally with the opposite half of the cerebellum. Hence in this case the large mass of transverse pontine fibres which chiefly form the middle cerebellar peduncle on the right side, and is in connexion with the larger right half of the cerebellum, is distinctly bigger than its fellow and displaces the anterior median groove of the pons to the left. It is interesting to note too that in relationship with the changes in the frontal and temporal lobes the temporo-pontine bundles situated in the dorso-lateral angle of the pons are somewhat wasted, and the fronto-pontine fibres situated mesio-ventrally are also wasted, but less so, in keeping therefore with the lesser affection of the main mass of the associated area of the frontal lobe. The right pyramidal bundles are much wasted and to a considerable extent broken up, more so than on the left side, by the transverse fibres of the pons.

Medulla.—Corresponding abnormalities obtain in the right half of the medulla where the pyramidal fibres are greatly diminished in number and some in size. The middle fillet is also smaller than its fellow, though the difference in bulk is not nearly so great as between the middle fillets higher up in the brain stem and notably in the mid-brain. Also, there is a distinct relative smallness on this side (right) of the nucleus lateralis, and of the inferior olive. The internal arcuate fibres are less numerous and also the fibræ comitantes quinti, though the spinal roots of the fifth nerve seem equally well developed. It is to be expected that the inferior olive, which is known to give origin to the system of afferent fibres (olivo-cerebellar) which cross the middle line and terminate chiefly in the opposite half of the cerebellum, should in this case be smaller on the right side because it is thus in connexion with the smaller half of the cerebellum (the left), but it is curious that the internal arcuate fibres should be more numerous on the left side of the medulla than on the right, because after decussating in the middle line they bend forwards (frontally), and to a great extent form the right middle fillet which is smaller than its fellow. It could not be

proved that the left posterior column nuclei from which the left internal arcuate fibres almost entirely take origin were larger (or smaller) than the right, yet the fact that the internal arcuate fibres were more numerous on the left side was not only apparent in the medulla, but similarly their homologues throughout the pons were also more numerous and better stained than those on the right. The bundle of Schütz, a network of fine fibres extending from the medulla to the cerebral peduncles and lying just dorsal to the twelfth and similar nuclei, is less evident on the right side than on the left. But, as would

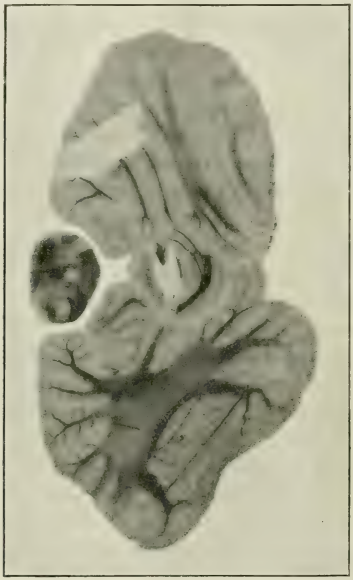

FIG. 8.

be anticipated, the right inferior cerebellar peduncle, formed chiefly by the junction of the right direct cerebellar tract with fibres coming from the larger left inferior olive and passing to the (larger) right half of the cerebellum, is larger on the right side than on the left. The remaining structures in the medulla seemed normal, and call for no comment.

Cerebellum.—The gross changes in the cerebellum are best realized

by a reference to the accompanying figure. The left half (i.e., the side opposite to the affected cerebrum) is much atrophied. On microscopic examination it is found that in the wasted area the cells of Purkinje are small in number and in size and their processes are much stunted, though here and there a few comparatively healthy cells may be seen. The granular layer, and slightly also the molecular layer, is thinner than in health and seem to be equally affected throughout their depth. An outer layer of round cells, two or three deep, immediately beneath the Purkinje cells, as found by Mott and Tredgold in a similar case, are not in this instance visible. The so-called cells of Golgi seem also distinctly diminished in number in the diseased area. The left nucleus dentatus is smaller and thinner in all dimensions than the right, and evidently associated with this is the fact that the left superior cerebellar peduncle which takes origin from it is smaller than its fellow. The nuclei globosus and emboliformis are slightly smaller and less rich in fibres than on the opposite side, and the left nucleus fastigii (*vel* tecti) is very distinctly smaller, and owing to a paucity of fibres and internuclear fibrillar network is paler and less well marked than on the right side.

The spinal cord.—The spinal cord is profoundly asymmetrical, and though both sides are affected, the left is much smaller than the right—the atrophy of the left side of the cord being in keeping with the arrested development of the right cerebrum and the left side of the cerebellum. The right half of the spinal cord presents no obvious diminution in size except in its anterior column. On microscopic examination it is seen that the right direct pyramidal tract and the left crossed pyramidal tract are much smaller than normal, the number of their fibres is much diminished, and, in addition, many of the fibres present show the ordinary signs of secondary degeneration and sclerosis. Moreover, these processes have not been limited to the above mentioned tracts, but involve also, though to a slighter extent, the deep, and more particularly the marginal, fibres of the left antero-lateral column. Similar slight changes are noticeable in the right crossed pyramidal tract and right antero-lateral column due to affection of some of the homolateral fibres. The posterior columns present no definite abnormalities, but the white fibres of the anterior commissure are in most sections decidedly more abundant on the *left* side than on the *right*. The left anterior column, which, by contrast with the atrophied right, seems almost larger than normal, is very well developed, and has displaced the median longitudinal fissure to the right. The

grey matter of the spinal cord is affected on the two sides, and that somewhat irregularly at different levels. The central canal, though not unduly dilated, is here and there multiple, so that two or three small canals lie in contact, each surrounded by the ordinary (healthy) columnar epithelium. The stress of change in the grey matter has naturally fallen on the anterior horns, and on the whole the left anterior horn is smaller, narrower, and more stunted than the right. This is especially seen in the cervical and lumbar enlargements. In a somewhat similar case as this, published by Mott and Tredgold, and previously quoted, the cells chiefly wasted were in the lateral and postero-external groups, but here, though these are to a large extent affected, and in some places practically absent, it is noteworthy that at certain levels the mesial groups, both anterior and posterior, as well as the central group of cells, are almost absent in the left anterior horn. The cells, mostly of smallish size and fusiform, of the lateral process to which Bruce has recently called attention, are well preserved at most levels, and better on the right than left. The columns of Clarke are little affected, but in the lower dorsal region their cells are larger and slightly more numerous on the right side. In all cases, however, these cells show a marginal accumulation of granules, with a peripherally displaced nucleus, that is to say, the ordinary structure of the Clarke-Stilling cells.

Remarks.

Such cases as described above have usually been known under the name of hemiatrophy of the brain or arrested development of the nervous system. All of them are probably due originally to a vascular lesion, either arterial or venous, occurring in fœtal or early life. The chief organic changes result from arrested development of certain parts of the central nervous system, whilst others continue to grow more or less as in health; hence the title of atrophy or hemiatrophy seems ill-chosen. On the other hand, as a result of arrested development and function in certain structures, others often far distant from the original lesion undergo secondary and tertiary atrophy, and these atrophies may give rise to the most striking organic changes or symptoms in a case, and therefore the term of arrested development of the nervous system or hemiagenesis suggests a distorted idea of the case. On the whole, it seems to the writer that the term microgyria, which involves no causal theory of the disease, and yet at once indicates the grossest change visible to the naked eye in such a case, is better than either of the other two in common usage.

Mott and Tredgold [2] state that these cases readily group themselves into two classes: (1) Cortical, in which the lesion is confined to the cortex or underlying white matter. (2) Basal, in which the original lesion is situated in the structures at the base of the brain; and they describe three cases as illustrating this. Though undoubtedly most of these cases can be thus conveniently grouped, there are occasionally others in which the original lesions may be both in the cortex and in the basal structures of the brain. Judging from the relative amount of disease in the cortical efferent systems and in the structures originating or terminating in the basal ganglia, the writer is of opinion that the case described above falls into an intermediate group of cases, partly cortical, partly basal.

Mott and Tredgold [3] state that "it has been conjectured that a descending thalamo-spinal path exists in man." By reference to two cases, including one of microgyria, and by a lucid deduction from facts. they proved the existence of such a path, and gave photographs of it, It may be of interest in this connexion that the writer, at a meeting of the Physiological Society at Cambridge in August, 1904, demonstrated specimens of the brains and cords of twelve monkeys in which he had caused degeneration in such a descending thalamo-spinal path by production of a localized lesion in the optic thalamus. This atrophy, or degeneration of the thalamo-spinal path, in the case described in this paper, is not (unlike that in the case described by Mott) obtrusive, because, firstly, the relative amount of disease in the thalamus is smaller; and, secondly, the amount of disease in the surrounding structures in the record is relatively greater.

It is not within the scope of this paper to enter into further detail, which, with organic changes in nearly every part of the central nervous system, would take one too far afield. The main fact that the right half of the brain is structurally and functionally chiefly associated with the left half of the cerebellum, and with both sides of the spinal cord, but chiefly the left, finds striking illustration in the present case, and further detail is given in the body of the paper.

REFERENCES.

[1] Mott, F. W. "The Progressive Evolution of the Structure and Functions of the Visual Cortex in Mammalia"—The Bowman Lecture, *Trans. Ophth. Soc.*, January, 1905, vol. 25, p. liii.

[2] Mott, F. W., and Tredgold, A. F. *Brain*, 1900, vol. xxiii, p. 239.

[3] *Ibid.*, p. 248.

A PSYCHOLOGICAL INTERPRETATION OF ESSENTIAL EPILEPSY.

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I.

As ordinarily considered to-day, epilepsy is recognized by loss of consciousness and a convulsion. Probably epilepsy as a disease has a more ancient lineage than any other nervous disorder. Throughout the ages the nature of epilepsy has been, and still is, the subject of surmise, conjecture and scientific study. The disorder has been ascribed to many varying factors. In the great majority of cases, however, no physical or structural causes may be found. These forms are spoken of as genuine, idiopathic or essential epilepsy. It is to the psychological nature of these cases especially that the interpretation in this short thesis is to be applied. Thus we have under consideration an apparently sound individual having epileptic attacks of apparent idiopathic or unknown origin, and whose essential epilepsy, therefore, may be considered in the nature of a life-reaction, comparable to a state of rage or anger as seen in bad-tempered individuals, or excessive emotionalism in the supersensitive.

It would appear that a full recognition of an epileptic constitution as such, independent of the deteriorating influence of excessive attacks, which only brings it more markedly in view, was not much recognized before 1860 or 1870. Falret, the elder, an observer of about that time, and Billod (1882), a pupil of Falret, as well as Kirchhoff, who wrote as late as 1890, believed that the peculiar mental make-up of so-called sane epileptics did not possess characteristics distinguishable from that of others in their particular station in life. It is interesting to note the view which a practical neurologist would have upon the presence of a particular and distinctive constitutional make-up, the essential contention of our thesis. Oppenheim, a neurologist of this type, states that the intellect of an epileptic may be "absolutely intact." He cites as examples Cæsar and Napoleon, but quickly adds that we see no such cases to-day. In another division of his work he states that sane

epileptics are "often excitable, suspicious and irascible," but hastens to offer an apology for these peculiarities, based upon the nature of the disorder from which they suffer. He finally adds, however, that a normal intellect becomes impaired by the fits.

As soon as the disorders of affect were recognized in psychiatry, independent of purely intellectual ones, a notation of the former began to appear in the literature of the psychology of epilepsy. Now and then one finds in the neurological literature, in contradiction to the psychiatric or psychologic view, an attempt to find an organic basis for the epileptic make-up, just as some authors strive to see many of the mental characteristics produced by the social restrictions which the interned epileptic endures as a result of his malady. Cramer (1912), while firm in the belief that epilepsy proceeds from cortical alterations, becomes confounded and unable to explain the constitutional make-up by such changes. Among the phenomena of latent epilepsy, Hartmann and di Gaspero (1914) believe that, long before the epileptic symptom complex, with its first undoubted paroxysmal phenomena, becomes manifest, an individual may show prodromal symptoms, the relationship of which, however, to epilepsy cannot be maintained with certainty. They cite many variations in affective life from the family histories of epileptics, and find they are hardly ever missing from such histories. Here belong the tendencies to abnormal, endogenous changes of temper, anxious fears and sudden depressions. Apart from the sequelæ and prodromata of an attack, the authors mention great exhaustibility, restlessness, anomalies of disposition, increased irritability, distrust, depression, anxious fears, tendency to violent impulses. In addition to the usual slight mental defects, the authors note a tendency to unreal ideas. There are also memory falsifications. The affective life as a rule shows great departures. There are transitory phobias, ecstasies, and, most common of all, irritable and depressive states. As many as 78 per cent. are said to suffer from these endogenous anomalies of disposition. As a rule consciousness is clear. Under the progressive changes seen in advanced cases of epilepsy, the authors refer to the deterioration. As a rule the subjects react abnormally to external impressions. The affective changes constitute that ethical depravity of the entire personality known as the epileptic character. We see here a morbidly augmented irritability of the temper, with a special tendency to outbreaks of anger. An egocentric contraction of the entire emotional life, pathological lying, &c., may develop.

Féré (1890) believed that the character and manner of epileptics can easily cause suspicion of the disease long before the convulsions appear. The emotional make-up is mobile and explosive; the former rests upon a foundation of "impotence and sadness."

Anyone acquainted with the well-formed peculiar mental characteristics of the frank epileptic in institutional life will easily recognize the classic description of the epileptic make-up as given by Pilcz (1904). In relating anything, the epileptic is never diverted and always arrives at a conclusion. He repeats the same story in the same language, and makes free use of commonplaces, proverbs and biblical texts. As a rule they are pietists, observe religious duties scrupulously. Their devotion is at times exaggeratedly sweet. They flatter by frequently prefixing "good" and "kind" in addressing others. In striking and characteristic contrast to the foregoing is the unusually marked irritability, sensitiveness and egotism. Hübner (1914) also mentions the inclination to piety on the one hand, which plays a great rôle in the thoughts of the epileptic, and on the other the strongly marked egotism which permits him to think only of himself and never of the rights of others.

In accord with the main tenets of the views given in the foregoing citations, one might quote a number of other writers such as Esquirol, Bischoff, Clouston, Ball, Russell Reynolds, Magnan, Howden, Romberg, and others. However, I think it may be proved, so far as opinions and observations of the chief investigators of our subject are concerned: (1) There is a more or less constant affective disturbance in all epileptics, sane as well as insane; that such defect is due to an inherent make-up of the psyche in which mainly an egocentricity and a highly sensitized feeling are given to the individual; and that from this constitutional make-up or alteration the ultimate deterioration of the psyche, intellectually as well as emotionally, is gradually developed, step by step, and if the state is not corrected that this finally and logically ends in real mental deterioration. (2) The epileptic alteration is seen to proceed from the mental make-up or constitution of the individual epileptic long before his malady reaches the convulsive stage, and that the one is but a further and final unfolding of the former.

A few years ago I undertook a study of the character make-up in a small series of essential epileptics *before* they had their first attack, and found these potential epileptic individuals had nothing wanting in the

complete picture of the character as seen in those suffering from a long enduring and severe epilepsy, though the character faults were not so glaring as in the frank epileptic; there was also a wide latitude in the quality and amount of defect encountered. It is interesting to compare the attitude of trained clinicians in epileptic colonies and physicians in private practice toward the epileptic individual. The former live more or less in intimate contact with their charges: they recognize their peculiar type—one scarcely detected by the outside physician. Hence, they base treatment largely upon the defects and capabilities of their charges, and grow less and less to depend upon the seizures as such, or to lay stress upon a pure drug or special organotherapy, a practice so common with the general practitioner; yet the position of the trained epileptologist, until recently, has been assumed by many to mean that he is unacquainted with the newer and ephemeral panaceas for this great disorder, and has arrived at therapeutic nihilism. The fact is, the colony physician has a broader view of the whole problem. The physician in private practice sees little of his patient's personality and individual reactions, and often only obtains a record of attacks and their allied symptoms upon which he may base his therapy. If the institutional physician received his patients in an earlier stage, he could probably considerably alter the pessimistic prognosis of epilepsy which now obtains. At present he receives not only the most badly deteriorated patients, but often those so fearfully handicapped by drug and surgical therapy that it takes months to rid the epileptic from these well meant, but misdirected, remedies, so called. It is to be regretted that the majority of text-books continue to state that sedatives are the chief, if not the sole, therapy for essential epilepsy.

II.

Inasmuch as the life reactions of the epileptic character are the distinguishing factors of the make-up, we may sketch the defects of maladjustment at the several epochs of life stress.

At birth the potential epileptic child frequently has periods of meaningless crying. This extra-irritability and sensitiveness is so pathological that it rarely fails of detection and record. For instance, one history states the child "was considered a nervous and irritable child from the day of its birth." A second "fretted continually at the contact of rough clothing," while a third "cried continuously the first

three months," although nothing physically abnormal could be detected. One infant nearly went into spasms at the sound of an air-brake. Still another had to be rocked continuously, and slept only during this care.

The next important sequence in such a character is its non-pliability in being taught nursery ethics—that is, obedience and proper daily deportment in the home. These behaviour defects are usually independent of purely intellectual and physical ones, and these infants sit up, creep, talk, and walk at the usual ages. Indeed, in not a few the physical and the intellectual development seem accelerated, and certainly are hyperactive. Extreme lability of mood is a frequent factor. One moment contented, and the next irritable beyond power of appeasement, is often noted. The consecutiveness of purpose in play and capacity to be amused is short, requiring the constant attention of the parent and varying appeals to the child's interest. Tantrum episodes even under the mildest discipline are most common in these difficult children. Their maladaptive defects are soon shown in their association with other children. They demand the play to be arranged to suit them; a game must be played a certain way; it must be continued or stopped as they direct. Their likes and dislikes are extreme. In the school under an impartial discipline these potential epileptics show the most obvious traits. The mood is inconstant, the interest and attention continually vary, and hence their more purely intellectual processes often show wide dissimilarities in evolution. Extremely brilliant in some subjects and quite ignorant in others is the general rule. Because of their extreme nervousness and inability to conform to school routine, many children fail physically and mentally, and cannot take on the normal school training. Some are privately tutored; others are exempted from school discipline and routine except for short periods. Such scholars often appear pale and haggard, the pupils of the eyes are dilated, and they grow lethargic and sullen, or sit day dreaming and yawn. They grow intolerant of the school, and often rebel at its exacting demands. Thus in infancy and early childhood the instinctive defect of the ego fails of proper sublimation; the adjustment to the social and physical environment is incomplete and unsatisfying, and, thrown back upon itself, in consequence there is a reinflation of self-importance and sensitiveness. Childhood, therefore, is a fruitful period for new and unbearable stresses, and, in consequence, epileptic attacks often occur in one predisposed.

The next stressful period is at puberty; here the dissolution of the oftentimes irksome home ties may release some from the galling exactions of home discipline, but most frequently, as in the departure for school with its exactions in deportment, we find the puberty adjustment to work and social demands increasingly onerous. The potential epileptic is not willing to take on a proper attitude of apprenticeship. He has the innate instability of the constitutional inferior, but the good-natured indifference of the latter is not his attitude; his feelings are easily hurt, he has ill-defined paranoid persecutions which cause him frequently to react with violence, insolence, and hatred. He has attacks of rages, sullen moods, and dispirited acquiescence in plans and undertakings. The lack of good-fellowship renders him incapable of co-operative teamwork. The potential epileptic admires co-operation and the doing of big things in the abstract, but the requirements of interdependence and subordination to the main purpose galls and irritates him beyond endurance. During adolescence he begins already to present an odd mixture of primary defects of instincts, together with a commencing deterioration of the higher capabilities of social adjustments known as habit deterioration. In part the latter is protective; that is, under the extreme stresses of this period he may ease off the stress by lowering or evading the exactions of precise behaviour and deportment. Thus, instead of presenting fits for a time, he may take on various types of dissipations. With no real or intimate friends, the potential epileptic holds himself aloof from the demands of the common sacrifice of self and the mutual dependence of social custom. He is a free lance, able and usually anxious to work his own will upon the world. If gifted with extraordinary intellectual endowments, he may succeed for a time and make satisfactory progress towards his goal, but just short of it his egotism and ambition are often fired to new and impossible ends; when the whole scheme seems to him within final consummation, some small trick of fortune adds the last burden of stress upon his mental and physical health, and the individual breaks into frank manifestations of his disorder.

Freed in greater part from the trammels of social concern and demand, the intellectual efforts of the potential epileptic work with less stress than the normal, but in the final and more advanced consummation of his task the social customs of the family, friends and society itself fail to add their small but modifying influence as a directing force to the effort. In

consequence new and unforeseen hindrances are added which in turn reduce the mental and physical invulnerability to the minimum, and at such defective periods some physical stress which at a more favourable time would be quite negligible appears as the precipitator of the frank disorder. Thus one learns to discount the common precipitating cause of the epileptic attack ; such obvious studies are but surface ploughing of the etiological field in epilepsy. One is apt to think that inasmuch as epilepsy is essentially a disease of early life the great majority are unmarried, because the state in itself bars marriage, but in the vast majority the reason is by no means so simple. It is really because the epileptic is rarely equipped in essential character make-up for marriage. Emotionally and sexually he rarely develops beyond the level of puberty and fails in capacity to attain adult love. Naturally this is to be expected in that the latter demands self-subordination and sacrifice, and above all a tenderness of feeling which is conspicuously lacking in the potential epileptic. In view of the foregoing it is evident that marriage increases demands socially as well as economically and makes not a few potential epileptics break out in attacks. At the threshold of life the vast majority break before or just at this period, which is the point of maximum stress. Almost all potential epileptics, long before their disorder becomes at all acutely manifest, show increasing slowness and a diminished capacity for sustained employment. They show extra fatigue and diminished interest, a partly protective mechanism. A tenacious, consistent, all-around emotional development in a life-work seems impossible to the vast majority. They work fairly well for a time, with plenty of emotional appeal, lavish praise and constant change, all essentially infantile traits of character, but in the end fail to do a thoroughly competent life-work.

III.

The muscular convulsion may be explained hypothetically. We know the convulsive part of the fit in its severer and cruder aspects is comparable to the impulsive movements of the infant. The impulsive fœtal movements begin about the twelfth week of gestation, hence the brain cannot be involved *per se* in their genesis ; further, it is known that brainless embryos possess impulsions. There is a short period just before birth in which the amniotic fluid and the uterine wall greatly inhibit the free play of these impulsive movements, but they

begin again with renewed activity in the newly born and are slowly inhibited by voluntary control at the end of the nursing period. We do not know just how the impulsive movements are incited further than to surmise that, being of the first, simplest and ontogenetic type of activities of the developing organism, their incitor is from motor centres of the lower order. In these latter structures are stored up a certain quantity of potential energy which is transformed into actual energy by the blood and lymph stream. With the increasing tissue growth and tension engendered thereby this energy finds its outlet in the random movements of the fœtus and the infant, and their exaggerated distorted presence is seen in the major convulsion of epileptics. Space prevents us from outlining more in detail the essential distinguishing characteristics of the impulsive from the instinctive, reflex and conscious or ideational movements of the infant; this has been done most carefully by Preyer and later correlated into a recent study by Canestrini. Suffice it to say the newer studies on the meaning of the convulsive part of the epileptic fit make careful analysis of all the impulsive movements of the nursling doubly necessary. It will then be found desirable to note their exact relationship in reference to the psycho-sexual development and its defects as shown in the infantilism of the epileptic.

As might be expected the number of the impulsive movements is not great. They may be schematized as those of stretching and bending the arms and legs in the newly born. The movements are sometimes so quick as to resemble the clonic convulsions of a fit. They may be slow, then fast and finally end in clonic movements. Even in healthy infants they may be so slow as to resemble the tetanoid spasm of a beginning focal seizure. Preyer speaks of the muscles involved in the impulsive acts as possessing such a slow, crawling movement that the acts present a striking resemblance to the extension and flexion of the limbs of animals waking from their winter sleep. Such animals, like sleeping children, seen even in the first half of the second year, make genuine fœtal movements which often look as though they were directed against some invisible resistance. This all suggests many of the striking impressions one gains in observing the convulsions of epileptics. Convulsive motions in the infantile impulsions are, however, not generally so frequent in sleep as slow contractions. The latter are frequently attended by spreading and bending of the fingers, which in turn become the rarer toward the end of the second year in all children of sound

nervous systems. All these impulsive movements, in the hands especially, are asymmetrical in outline. What are some of the depressors and incitors of these impulsive movements? Deep sleep reduces them to the minimum. Satiation by food greatly curtails them. On the other hand, a duplication of the intra-uterine state by the use of the warm bath encourages them. The movements are then usually slow and rather rhythmic and graceful. One may even see in them the beginning of an expression of pleasure. The face may join in the picture of contentment with slow asymmetric contortions, which semblance has an odd mixture of pleasure with more than a hint of displeasure. The greater part of the impulsions, however, are purposeless, senseless and asymmetric and are found over the entire body from the first day of birth. Writhing and twisting of the body are also frequent accompaniments to the movements of the face and extremities. Just as the infant sinks into deep sleep these impulsive movements slow down and the body usually comes to a state of rest in the fœtal position. The fœtal posture in the legs is kept up longer in advancing child life than that of the head and upper extremities. Many writers have called attention to the fact that no one could consciously duplicate these acts. Then, too, one is strikingly impressed that the infant and the epileptic alike are little fatigued by these most intense and persistent impulsions, which speaks strongly for the unconscious motivation in both their activities. Probably in both subjects the fund of reserve energy being so limited in scope is greater than that of the normal adult as ordinarily expressed in his daily activities. Biologically speaking, we know that the essential vital energy of an individual is probably at its maximum at birth.

We are justified in considering the essential nucleus of the epileptic fit an infantile unconscious striving of displeasure-pleasure pursuit ending in the final goal of a return to infancy, attended by a loss of consciousness and a convulsion; that the convulsion is made up of and flows out of the general striving of the fœtal and infantile tissues as expressed through the lower spinal centres in inducing simple and crude combinations of impulsive movements; that a study of the degree of development of unconscious infantile strivings in the emotional instincts, the desire for an infantile state of omniscience, are paralleled by the kind and character of impulsive movements found in this infantile period of neuromuscular development. Therefore the two main settings in the epileptic fit, unconsciousness and convulsion, are psychical and physical

correlates; lastly, that epilepsy in its essential pathogenesis is an error or arrest in this fundamental elaboration or development of the emotional life.

Having schematized the dynamic mechanism of the epileptic fit, we may note that the essential pathogenesis of the disorder as a whole is still to be attacked. Whether the latter rests upon an inheritance of certain psychic traits alone or whether there are certain somatic structural anomalies which do not permit proper emotional and instinctive development into normal adult life one cannot say. I believe such studies, however, narrow the gap between such causes and their psycho-physical expression in epilepsy; and finally, such observations must be of greatest aid in classifying the recoverable epileptics from the irrecoverable ones. It also points the way by which may advance our therapeutics of the disease along the broadest biological lines of educational and moral treatment. In this connexion one may note that MacCurdy has tentatively formulated the idea that the sudden loss of consciousness in epileptics liberates a muscular anarchy of "clotted mass of movement" of many different lower levels or physiologically controlled centres in the brain and spinal cord, and that the convulsive part of the fit is a released neuromuscular mechanism or series of mechanisms entirely secondary to the loss of consciousness, the main defect of the epileptic state. He holds the same fundamental postulate as to the psychological meaning of the epileptic fit as we have formulated it.

IV.

Given the inherent defect of make-up, it is easy to comprehend how all forms of undue physical and mental stress may operate deleteriously upon the epileptic. The gradations of epileptic reactions vary from day-dreaming, lethargies, petulance, sullenness and outbursts of impatience, until there succeeds a series of minor attacks or a severe major convulsion, when the lowering skies in the epileptic's life are dispelled for a time until the stresses again accumulate to an explosive level. So long as normal consciousness is maintained the stress may work its evil consequence in ways well known to all. The epileptic reaction from its mildest to its severest manifestation is really a protection, for it obliterates reality and reduces the subject to the lowest level of organic response, that of a comatose state. Hence the fit is really a protective mechanism, psychologically considered at least. It withdraws or reduces the

subject's attachment and adjustments to reality. It dispels an intolerable demand and the epileptic retreats to a state of harmony and peace. In the retreat after incomplete attacks we frequently encounter unconscious strivings and conflicts that have baffled the subject. So exogenous causes, physical and mental in character, slight as they may seem to be, precipitate a conflict which fires the gun, and a series of conflicts of different levels in the unconscious are exposed until after the severe major attack the subject is reduced to the lowest level, comparable to earliest infantile life. When this hypothesis was enunciated a few years ago it was as yet uncorroborated by exact data, but since that time innumerable studies bearing out every contention of the mechanism have been deduced. Thus we find in the mild and transitory deliria of the automatic phase after petit mal attacks the subject may say or do certain things which may be pieced together and minutely analysed. Like the mental content in maniacal states or drug and fever deliria, these spontaneous productions have to do with the conflicts of everyday life. Then appear the successive deeper levels of emotional strivings and conflicts. For instance, at first the epileptic attempts to rid himself of an onerous task or unpleasant companions; sexual strivings are uncovered and in the deepest level he has made a retreat to the home, is in the cradle or the mother's arms, &c.

The view here expressed in reference to the psychology of epilepsy is not essentially new either in principle or application of treatment. It but gives us a more rational understanding of the nature and mechanism of the disorder itself and renders the plan of handling and treating the essential disorder more understandable and practicable. One can easily comprehend that the point of attack in the treatment of such a disorder rests upon an effort toward training out the constitutional defects of personality and teaching these patients to make a better adjustment to life.

We therefore must conclude that essential epilepsy as regards causation is dependent upon a primary congenital defect or inheritable defective instincts of natural and healthful adaptations to reality, the epileptic make-up or constitution, and that at successive periods in life, when emotional and physical stresses of an intensive character are encountered, the individual so constituted has epileptic reactions such as fits, disturbances of temper, lethargies, and various psychic phenomena. The fit is essentially a regressive phenomenon, a flight from

undue stress into unconsciousness, the deeper manifestations of which, the convulsive phenomena, are superadded. By analysis of the states of automatism of epilepsy, by the conscious analysis and the dream states, one gets to know the depth of regression and the infantility or crudity of the essential instincts of the individual epileptic. In consequence the future lines of treatment heretofore empirically undertaken by reducing the life stresses is now made much more patent by our exposition of the instinctive defects: that the proper treatment of epilepsy is an intensive and persistent educational training in its broadest and deepest sense, together with the correction of such mental and physical disorders as are remediable by more conscious methods, as occupation, educative play and healthful interests and pursuits.

REFERENCES TO FULLER STUDIES BY THE AUTHOR OF THIS PAPER.

"Nature and Pathogenesis of Epilepsy," *New York Med. Journ.*, 1915, vol. ci, pp. 385, 442, 515, 567, 623.

"A Personality Study of the Epileptic Constitution," *Am. Journ. Med. Sci.*, 1914, vol. cxlviii, p. 729.

"Clinical Studies in Epilepsy," *Psych. Bull.*, *N.Y.*, 1917, vol. xi, p. 21.

"The Psychological and Therapeutic Value of Studying Mental Content during and following Epileptic Attacks," *New York Med. Journ.*, 1917, vol. cvi, p. 677.

"A Further Study of Mental Content in Epilepsy," *Psych. Bull.*, *N.Y.*, 1917, vol. xi, p. 483.

"The True Epileptic," *New York Med. Journ.*, 1918, vol. cvii, p. 817.

"A Study of Certain Aspects of Epilepsy compared with the Emotional Life and Impulsive Movements of the Infant," *Interstate Med. Journ.*, 1915, vol. xxii, p. 969.

"Treatment of the Epileptic, based on a Study of the Fundamental Make-up," *Journ. Amer. Med. Assoc.*, 1918, vol. lxx, p. 357.

THE PSYCHOGALVANIC REFLEX: A REVIEW.

BY E. PRIDEAUX.

(*From work carried out at Cambridge for the Medical Research Committee.*)

Waller [45], during his investigations on the electrotonic changes of excitability of the motor and sensory nerves of man in 1880, noticed irregular galvanometric deflections, which at that time he could only explain as being due to slight alterations of contact between the skin and the electrodes caused by muscular movement. Féré [7] was the first to point out in 1888 that an emotional reaction was accompanied by electrical changes in the human body, demonstrable by galvanometric deflections, when the resistance of the body to a weak electric current was being investigated; he noted that these changes were correlated with the alterations in volume of the limbs as shown by the plethysmograph, but reported that the galvanometric deflections had been insignificant in the normal persons he had experimented upon, and were only established for hysterical patients. In Féré's opinion the changes were due to a lowering of the resistance of the body under the influence of emotional states.

In the same year, independently of Féré, Tarchanoff [42], working on the electromotive changes in the body, and using no external current, showed that the phenomenon was a normal one, and occurred in all healthy individuals, when two non-polarizable electrodes attached to asymmetrical parts of the body were connected up with a galvanometer. Tarchanoff concluded that "the galvanic phenomena observed in the skin, which are concomitant with different kinds of nervous activity, should be considered as due to an active state of the sweat glands, which at this time produce a secretory current."

The work of Féré and Tarchanoff remained for several years unnoticed, and it was not until E. K. Müller [24], an electrical engineer without any knowledge of the previous work, rediscovered Féré's phenomenon in 1904, and communicated his results to Veraguth [43], who gave the name "psycho-physical galvanic reflex" to the phenomenon, that any methodical research was carried out. Although Veraguth's term, now shortened to "psychogalvanic reflex," has come to be used indiscriminately for all electrical changes shown by the

galvanometer due to psychophysiological processes, it is necessary, in order to avoid the confusion in which some investigators of this subject have entangled themselves, to deal separately with the phenomena as reported by the original observers: (1) Galvanometric deflections due to production of some electromotive force when no external current is used (phenomenon of Tarchanoff); and (2) Galvanometric deflections due to apparent change of resistance when a weak electric current is passing through the body (phenomenon of Féré).

Apparatus employed to demonstrate the Reflex.

(1) *When no external current is used.*—Tarchanoff [42] employed non-polarizable clay electrodes attached to the skin by means of hygroscopic pads saturated with saline solution. These were connected with a Meissner and Meyerstein galvanometer. The electrodes were applied at different times to asymmetrical parts of the body such as the hands and fingers, feet and toes, face and back, whilst the subject was kept at complete rest. Experimenting in this way he found that psychic stimuli, real and imaginary, provoked galvanometric deflections. Later, investigators have followed this method with but slight modifications. Sticker [41] repeated Tarchanoff's experiments in 1897, and Sommer [39] in 1902. Fürstenau [8] experimented on the physical nature of the phenomena and the effects of different electrodes. Sidis and Nelson [38] relied chiefly on platinum hypodermic electrodes, used a d'Arsonval galvanometer ("sensibility 225 megohms"), and took photographic records of the deflections; they showed that even from symmetrical parts of the body, in the case of the two hands, a galvanic reflex was produced. Gregor and Loewe [15] and Philippson and Menzerath [30] employed an Einthoven's galvanometer with photographic recording apparatus, the former relying chiefly on Ostwald's electrodes, and the latter on Einthoven's zinc sulphate electrodes.

Radecki [33], using zinc electrodes, got similar results with Lippmann's capillary electrometer, and found that the electromotive force produced was about 0·005 volt.

(2) *When an external current is used.*—Féré did not give any particulars of his method. The usual technique of most of the investigators is a modification of that employed by Veraguth. He introduced a d'Arsonval mirror galvanometer with a shunt into a circuit, through which a feeble electric current from one or two Leclanché cells was being conducted through the body, the places of entrance and exit of

the current being the palms of the hands, and the electrodes being nickel-plated brass cylinders. He measured the deflections of the galvanometer on a horizontal celluloid scale, on which the light from the mirror registered its movements. A similar technique, with the addition of a device for recording the deflections by hand on a kymograph, was adopted by Peterson and Jung [27, 28], using copper plates as electrodes, Ricksher [34] with brass plates, and Radecki [33] with zinc electrodes. Sidis and Kalmus [37], and also H. Müller [25], used liquid electrodes—glass vessels filled with concentrated saline solution, in which large copper electrodes of about 500 cm. area were permanently placed, and in which the hands or the feet could conveniently rest. Gregor and Loewe [15] were the first to take systematic measurements of the deflections in terms of apparent resistance, and compare it with the resistance of the body by means of a Wheatstone bridge. Waller [47, 49] independently has adopted this method as the most convenient for accurately determining the magnitude of the deflections in terms of ohmic resistance, or, reciprocally, of conductance, introducing also into the circuit a potentiometer delivering one-tenth of the E.M.F. of a Leclanché cell for purposes of calibration, by means of which the deflection can also be directly measured and expressed in terms of voltage; he passes his main current from two Leclanché cells through one hand, and applies the electrodes, usually zinc discs covered by chamois leather moistened with 0·6 per cent. saline solution—one on the dorsum and the other on the palm of the hand. By this method he has shown that the reaction expressed in terms of voltage may be 0·5 volt.

Phenomena of the Reflex.

The only fact about which there is general agreement amongst the investigators on this subject is that the phenomena of the reflex have been definitely established, both with and without the use of an external current.

(1) *Galvanometric deflections due to psychical causes.*—Any stimulus giving rise to an emotion, such as the unexpected ringing of a bell, flashing a light, a pin-prick or a burn, after a certain latent period will cause a deflection which is proportional to the subjective state aroused. An actual stimulus is not necessary, the expectation of the stimulus is sufficient, and it is only necessary for the experimenter to walk behind the chair of the subject for a deflection to be produced. The threat of

a stimulus often provokes a greater reaction than the stimulus itself. Also calling up memories of painful experiences, or referring to questions concerning which the subject may have pin-pricks of conscience, will give a deflection.

(2) *Galvanometric deflections due to physical causes.*—Alterations in contact between the skin and the electrodes will cause deflections, but these are easily recognized. When using liquid electrodes, this deflection is always in an opposite direction, due to withdrawal of the hands or feet from the solution: when using metal electrodes, firm pressure on them can produce a very slight deflection in the direction of the emotional reaction, but muscular movement causing diminution of contact will produce a deflection in an opposite direction. These deflections can be distinguished by the fact that they take place immediately, and are not characterized by a long latent period.

(3) *Galvanometric deflections due to physiological causes.*—A deep inspiration, cough, yawn or sigh will produce a large deflection, whilst ordinary respiratory movements will not visibly affect the galvanometer. These deflections have a latent period of the same order as the emotive deflections.

(4) *Latent period.*—The latent period varies in different people, and in the same people at different times. It is of an average duration of two to three seconds. The periods recorded by different observers vary slightly, as some—Veraguth [43], Knauer [19]—make it longer, having measured the time from the commencement of the stimulus to the summit of the reaction, instead of to the commencement of the reaction, as is now generally done.

(5) *After-discharge effect.*—The reaction provoked by the excitation persists for some time. This time varies considerably in different persons; it may last only ten to twenty seconds, or it may persist for a few minutes.

(6) *Fatigue of reflex.*—After a stimulus has been repeated a few times it begins to lose its emotive effect, and very little reaction then occurs. It cannot be regarded as a true reflex fatigue. After a subject has been experimented upon for some time and has become familiar with the procedure, he may give no reactions until some new and unexpected stimulus catches him unawares.

(7) *Diminution of reflex as result of fatigue.*—The same subject will react differently to the same stimuli at different times of the day, and at the same times on different days, according as he feels in good or bad health. Physical or psychical fatigue diminishes the reflex.

Other important phenomena are that, when an external current is used, only the palms of the hands and the soles of the feet give a visible reaction, and that when no current is used the best reaction is given by a combination of left hand and right elbow. (Philippson and Menzerath [30].) When an external current is used there is a slow, constant deflection, indicating an apparent gradual increase in resistance when the electrodes are attached to the skin of the palms of the hands or soles of the feet, and an apparent decrease when they are attached to the skin of the forearms or legs. The apparent gradual increase in resistance was called by Veraguth "the curve of rest," and is now known to be due to a polarization effect.

Nature of the Reflex as a Physical Problem.

All investigators of the psychogalvanic reflex, having satisfied themselves of its reality, have admitted that the nature and causation of the phenomenon is an extremely complex one, and for the most part have only been able to put forward hypotheses to account for it.

In the case of deflections produced by emotional reaction when there is no battery in the circuit, the problem resolves itself into a physiological one to explain the causes for change of potential between the two electrodes.

But the physical problem is more complex when an external current is used, and great discussion has centred round the question as to whether the galvanic deflections are due to change of resistance or to the production of an electromotive force.

Féré's [7] view that the phenomenon was due to a lowering of the resistance of the body was tacitly assumed to be correct, and was upheld by Veraguth [43], who states: "The psychogalvanic phenomenon is an expression of the change of resistance of the skin under the influence of psychic phenomena." This explanation was accepted by Peterson and Jung [28], Ricksher and Jung [34], and Peterson and Scripture [29], without any definite experiments from this point of view. It was not contested until Sidis and Kalmus [37] reported that the phenomenon was definitely not of the nature of a resistance, but was essentially of the nature of an electromotive force.

Thus Sidis [36] states: "All our experiments prove incontestibly that the galvanic phenomenon is due to an electromotive force which is muscular in origin." Sidis's technique must therefore be examined in some detail. Sidis only employed [37] "a single cell giving a con-

stant E.M.F. of about one volt, which was sometimes replaced by a thermo-element giving only a few millivolts, and sometimes entirely removed from the circuit." But unfortunately he does not indicate what strength of current he used in the different experiments, beyond the fact that in most of the experiments on which he relies for his evidence he used no cell at all, and was therefore only dealing with an electromotive force. The strength of the current used in eliciting the reflex is however an important factor, and seems to have been ignored by previous investigators. I have shown in experiments, not yet published, that it is possible to diminish the reaction *pari passu* with the diminution of the current, and practically to abolish the reflex altogether, except for the slight one due to electromotive force, by reducing the current sufficiently. Therefore, if Sidis used a current of only a few millivolts, he would probably get a reaction due to electromotive force only, and not to change of resistance.

Secondly, Sidis, referring only once in all his experiments [37] to the latent period, which he found to be variable, but of the order of magnitude of a few seconds, confesses that "no attempt was made to study accurately such latent periods," and he omits to indicate what the latent periods were in his crucial experiments, nor had he any photographic records for this part of his work. As investigators have come to regard the latent period of an average of two to three seconds following the stimulus as the direct proof of a genuine galvanic reflex, and we know that muscular movement can produce a galvanic deflection, there is no *prima facie* evidence that Sidis in some of his experiments was investigating the psychogalvanic reflex at all. Thirdly, to complete our criticism of this part of Sidis's work, it is well recognized that the galvanic reaction following muscular movement occurs immediately after the stimulus, and is, when using liquid electrodes, in an opposite direction to that caused by the emotional reaction. Moreover, it has been clearly shown by Waller [48] that large reactions are obtained without any muscular movement visible on a myograph of sufficient delicacy to indicate the movements of the pulse. Sidis's theory that the phenomenon is due to an electromotive force of muscular origin can therefore be disregarded. Albrecht [3] also held the view that there were two factors in the causation of the phenomenon, and that of these the electromotive force was the more important.

That the phenomenon is due to an apparent diminution of the resistance of the body or skin, and not to the production of an increased electromotive force within the body, is evident from the following experiments:—

(1) As was pointed out by Radecki [33] and others, the deflection remains the same, and is in the same direction when the electrodes themselves are changed from one hand to the other. If the deflection was due to an electromotive force, then under these conditions the direction of the deflection would be reversed.

(2) Waller [48], using a current from two Leclanché cells (2·8 volt), has shown that the deflections may be of high apparent voltage (0·5 volt and above), whereas Radecki [33], using Lippmann's capillary electrometer, showed that the reactions due to electromotive force only averaged 0·005 volt.

(3) Waller [48] has shown that "during a persistent emotive deflection of high voltage, a superadded deflection, caused by throwing in an additional calibrating deflection, is augmented in proportion with the augmented conductivity."

(4) The experiment I have recorded above, namely, reducing the current sufficiently to abolish the reflex. If the reaction was due to electromotive force, then this procedure should increase the reaction instead of decreasing it.

Having decided that the phenomenon in the presence of an external current is due to an apparent diminution of resistance, the next question is whether the apparent resistance is one of the body or of the skin.

The determination of the resistance of the body to an electric current is a complex problem, for it depends on the degree of polarization and the number and nature of the ions which enter the tissues. We know, from the results of electrotherapy by ionization, that the glandular orifices are the probable channels by which the ions and the electric current penetrate the body. Leduc [20] has shown that with the same electrodes we get varying resistances with different active ions, and Girard [13] has shown that the permeability of the skin is not the same even to the same ions, as the skin and all the membranes are susceptible of giving rise to polarization. Any experiments on the resistance of the body must therefore take into consideration these polarization effects. Weiss [53] has devised an arrangement for measuring this polarization, and found a potential of 0·2 to 0·25 volt due to polarization of the tissues of the body. It is probable that this polarization effect varies considerably in different persons and in different parts of the body, and our psychogalvanic reflex experiments must make us suspect that it is at any rate often much higher in the palms of the hands and the soles of the feet. Gildemeister [10] claims that it is

possible to produce a contrary electromotive force due to polarization effects of two volts in the frog and of six volts in man.

With an alternating current the phenomena due to the polarization effects are reduced to a minimum. It has recently been shown by Gildemeister [12] that the resistance of the body varies according to the method of measurement. With the constant current the resistance depends on the duration and intensity of the current; with alternating currents the resistance is smaller the greater the frequency. After the removal of the skin the variations are smaller, whilst observations on the skin alone show the fluctuations just as the intact body does. It was pointed out by Aebly [2], working under Zangger, that the resistance of the body by Kohlrausch's method remains the same whether or not a current was passing through it, and that the resistance of the body to the alternating current does not alter under the influence of emotional reaction. By analogous experiments Gildemeister [9, 11] demonstrated the presence of the psychogalvanic reflex without any alteration in ohmic resistance, and Schwartz [35] got identical results in the frog.

It is now obvious why a diminution in the strength of the current reduces, and may if sufficient practically abolish the large reaction usually obtained when using a current of about 2 volts, for alterations of the current will affect the polarization of the skin.

We can, therefore, definitely exclude bodily resistance as a cause, and point to the polarization of the skin as the chief factor in the production of the reflex.

Knauer [19], in his criticism of Veraguth's book, "Das psychogalvanische Reflex-phänomen," got very near to this view, but it was H. Müller [25], and Aebly [2], who first drew attention to the phenomena of polarization and recognized that the reflex was a polarization effect; they held that the increase in conductivity of the body was due to a diminution of the electromotive force of polarization, and this view was upheld by Piéron [31], and in part by Gregor and Loewe [15]. The polarization effect then might be due to a diminution of the electromotive forces of polarization, as held by Müller, Aebly, and Piéron, or to a diminution of the resistance set up by the membrane where polarization occurs. Waller [48] has settled this point by the experiment here quoted. "If a potentiometer is put in series with the subject in the fourth arm of the bridge, we shall obtain indications that will enable us to decide between the two alternatives—electromotive force increased or resistance decreased. If the former, a known E.M.F. from the potentiometer should give the same deflection before and during an

emotive change. If the latter, the potentiometer deflection should be increased during the emotive change. And in point of fact this second alternative is what actually occurs; whenever I have measured the resistance in ohms before and during emotive excitement, I have found that the potentiometer deflection was inversely proportional with the resistance." We must conclude, therefore, that the psychogalvanic reflex in the presence of an external current is brought about by a diminution of the resistance of the skin due to the effect of polarization, and that this polarization is influenced by an emotional reaction.

Physiological Processes concerned in the Causation of the Reflex.

The physiological processes that will have to be considered as being possibly concerned in the causation of the reflex, both as causes for the change of potential between the electrodes when using no current, and as causes for the changes in the polarization of the skin in the presence of a current, are: (1) Unconscious muscular movement; (2) changes in the capillary circulation; (3) respiratory changes; (4) secretory activity of the skin; (5) nervous excitation through the intermediary of the cerebrospinal or automatic nervous system; (6) biochemical changes in the skin, or to a combination of these. But slight changes sufficient to cause a difference of potential, such as currents of action due to unconscious muscular movement or secretory activity, which may be the concomitants of emotion, are not sufficient to account for the large changes obtained when a current is being used.

(*a*) *Reflex due to production of an electromotive force.*—Investigators who have given theories to account for the changes of potential are Tarchanoff [42], Sticker [41], Sommer [39], Sidis and Nelson [38], Radecki [33], and Philippson and Menzerath [30].

Tarchanoff relied entirely on the activity of the sweat glands to account for the phenomenon, and explained the galvanometric deflection as being due to a secretory current. He showed that by placing non-polarizable electrodes on different parts of the body the area most rich in sweat glands became negative to the less glandular area. This question of the activity of the sweat glands must be taken up later when dealing with the processes concerned in the presence of a current.

Sticker repeated Tarchanoff's experiments, and, whilst not opposing his secretion theory, held that the changes in capillary circulation also played an important part; he based his view on the work done by

Hallion and Comte [16] on the influence of emotion on capillary circulation. Radecki's experiments support Sticker's theory; his conclusions are that the reactions are due to changes in the circulatory and secretory systems; that the changes of potential result from discharge of electricity which takes place during glandular activity, and that these changes depend directly on secretory and excretory processes, and indirectly on the capillary circulation. Though it cannot be denied that the above processes do cause changes of potential, there are serious objections to considering any of them as the specific cause of the psychogalvanic phenomenon. As Sidis [37] has shown, when using no current, exclusion of the skin by the use of hypodermic needle electrodes, or covering the skin with shellac and paraffin, leaving only the nails exposed, does not prevent the occurrence of a galvanometric deflection. Also exclusion of the circulation, in man by means of an Esmarch's bandage and in the frog by ligation of the arteries supplying the limb, does not materially affect the reaction. Sidis and Nelson [38], on the other hand, having excluded the skin and circulation, claim that the phenomenon is exclusively muscular in origin. They have recorded some very careful experiments from this point of view, and showed the presence of galvanometric deflections due to muscular contraction when the other causes were excluded. But the difficulty in accepting Sidis's conclusion is that these experiments were done on animals; that they do not mention anything at all about the latent periods, nor is it possible to deduce them from their records, and that they did not try any experiments in man from the point of view of excluding muscular contraction and leaving the skin as a possible factor. They relied on the fact that the deflection was abolished in the curarized frog. It is possible that they were in reality investigating the currents of action due to muscular contraction, and were only repeating the classical experiments of Du Bois Reymond [6]. The most recent work has been done by Philippson and Menzerath [30], who conclude as the result of their experiments that the origin of the difference of potential causing the galvanometric deflections during emotional excitement cannot be attributed to any one specific cause. They hold that it is due to the following combination of causes: (1) Exclusively cutaneous by changes in the activity of the sweat glands or in the chemical products secreted; (2) musculocutaneous when the right hand becomes positive to the left, which phenomenon they explain after Du Bois Reymond [6] as being due to the distension of the skin following involuntary contraction of the right hand; (3) exclusively

muscular. They also confirmed Tarchanoff's [42] statement that there is no correlation between the reflex and respiration, for the pneumogramme and the electrogramme do not show any parallel. It would seem *a priori* that the same changes in the skin which will account for the reflex in the presence of a current would also set up a difference of potential and produce the electromotive force reflex. The evidence to be put forward as the result of experiments with an external current tend to prove that none of these causes can be regarded as the exclusive cause of the reflex, and at present the problem is still unsolved.

(*b*) *Reflex due to change of resistance.*—Muscular movement can be definitely excluded by the experiments already cited.

The effects of circulation as a specific cause may also be excluded, for the reaction is not appreciably affected by the application of an Esmarch's bandage, as originally shown by Veraguth [43]. In spite of these experiments, Radecki [33] still maintains this view. He holds that the reflex is due to a vasodilatation provoked by a psychic excitation, and that the changes in the circulation cause an increase in gaseous exchange in the organism and thereby increase the conductivity. It is true that vaso-dilatation may cause a reaction in the same way that a forced inspiration will, but it is not the cause of the galvanic reflex.

The influence of the respiration deserves more attention than has yet been given to it. A deep inspiration, a cough or a yawn produce a most marked effect, often greater than that produced by emotion, whilst ordinary respiratory movements do not affect it. At first sight it would seem that here we might have an explanation of the phenomenon; but in an extensive study on this question, Peterson and Jung [28] have shown that there is no constant relation between galvanometric and pneumographic curves. They conclude: "When the emotions are very labile and show the most marked excursions in the galvanometer curve, the respiratory curve is often regular and even. On the other hand, where the galvanometer curve is marked by little fluctuation, or even by none, there will often be most decided variations in the pneumographic curve." These authors, referring to the rise in the galvanometric wave due to inspiration and coughing, state: "We are inclined to think that this rise may also be psychic, that is emotional. Certainly, in the curve we observe exhaustion by repetition of the command to cough or breathe deeply, as in the case of other analogous stimuli." It would seem more likely that the exhaustion in these experiments, which is, however, slight after fifteen deep inspirations and fourteen coughs, is

due to physical fatigue, and that the change in polarization of the skin produced by a deep inspiration is a purely physiological effect. If this is the case, then the changes in the skin due to a deep inspiration or coughing are analogous with the changes produced by emotion, but they are independent of each other. However, a study of the biochemical changes in the skin due to forced respiration might possibly give us the key to the psychogalvanic phenomena.

The theory which has been most strongly supported is that the phenomenon is due to alterations in secretory activity. Veraguth [43] supposed that the seat of the change of conductivity was the skin, and thought that this conductivity changed, for the following reasons: "The cells of the body, like colloids surrounded with a membrane, ought to show, each in its own particular way, a specific obstacle to migration of the ions. This way varies in the same cells, both according to general tone and according to the alterations in their state brought about by excitation." He excludes participation of the perspiration itself, for the results were similar in hands made dry with formalin, but he does not exclude secretory activity. He quotes one case in which he placed a belladonna plaster on his subject for three hours, then took it off and rubbed the hand with liquid extract of belladonna, and found that the psychogalvanic reflex was diminished.

Peterson and Jung [28] attribute the phenomenon to a secretory activity. "It would seem that the sweat glandular system is the chief factor in the production of this electric phenomenon, inducing on the one hand under the influence of nervous irritation a measurable current and on the other hand altering the conductivity of the current. Change in resistance is brought about either by saturation of the epidermis with sweat or by simple filling of the sweat gland canals, or perhaps also by intracellular stimulation; all of the factors may be associated." This view has been very generally accepted. Gildemeister [9], and more recently Philippson and Menzerath [30], have accepted this explanation, relying apparently on the experiments of Leva [21].

Leva found that in ten subjects a subcutaneous injection of 1 mgrm. of atropine sulphate entirely abolished the reflex; that directly after the injection, and for the next ten to fifteen minutes, the galvanometer deflection was of the usual order, but that after fifteen to twenty-five minutes it diminished rapidly, and after about thirty minutes even the strongest stimulus gave no reflex. The results of Waller's experiments from this point of view [50] are directly opposed to those of Leva. Waller states: "I have taken great pains to satisfy myself about the

action of atropine upon the emotive response, and have failed to find any evidence that it possesses any action at all upon the response." This view is supported by the experiments of Markbreiter, working in collaboration with Waller [22]. In one of these experiments, even after an injection of 2 mgrm. of atropine sulphate into the palm of one hand, when the usual pharmacological action was marked, no effect was produced on the galvanic reflex, and equal responses of the usual order were obtained from both hands. The inconsistency in the results of Veraguth, Leva and Waller seems difficult to explain. Neither Veraguth nor Leva give any photographic records of their experiments, nor any detailed accounts of their technique. Certainly in Veraguth's experiments the electrophysiological conditions did not remain constant during the test. It is possible that Leva, who used non-polarizable electrodes, was only dealing with a deflection due to a small electromotive force, caused by a current of action due to secretion, the nature of which was determined by Hermann [17] in his experiments on the forearm. In this case Leva was only repeating experiments analogous with that of Hermann [18], who showed that atropine suppressed the ascending current due to secretory activity, which was normally produced by excitation of the sciatic nerve in the curarized cat.

Further confirmation of Waller's atropine experiments is afforded by his results with pilocarpine and the calcium chloride capsule experiments, devised by him [46] for the measurement of insensible perspiration. He has demonstrated [50] that an increased emotion, as shown by the galvanometer, is not necessarily accompanied by an increased secretion, and in fact in the four experiments on the same subject which he quotes the contrary was the case, and an increased emotive deflection was accompanied by a decrease of secretion. Waller concludes: "While it is no doubt true that the deflection is due to diminished resistance, and that perspiration causes diminished resistance, it does not follow logically that the deflection is due to perspiration, since there are many occasions on which the emotive fall of resistance occurs without augmentation of either sensible or insensible perspiration." Waller's evidence is so strong that we must conclude that the change in polarization, and consequently the psychogalvanic reflex, does not depend on the secretory activity of the skin.

There remain therefore only the influence of nervous excitation, through either the cerebrospinal or autonomic nervous system, and changes in the biochemical processes in the skin dependent on them, to account for our reflex. Very little work has been done from these points of view.

V. J. Müller [26], in researches on the *Macacus cynomolgus*, found that the reflex was suppressed by perineural injection of an anæsthetic round the nerves supplying the palmar surfaces of the upper limbs, but that in man the reflex was not suppressed, but only diminished. Section of the nervous trunk gave an immediate suppression, and this suppression lasted while the electrodes were in contact with the palmar surfaces, but at the end of some time the dorsal regions, which did not previously give reactions, then gave them. This is not explainable, and has not been followed up by further work to verify this result. Sticker [41], working only on Tarchanoff's phenomenon, noted that the reaction occurred in both anæsthetic and analgesic skin areas, organic and functional, but there is not sufficient information on this point in regard to Féré's phenomenon. Any further consideration of the physiological nature of the reflex must at the present time be hypothetical. Waller's view is that the emotive phenomena belong to nutritional or trophic changes in the course of metabolism, that "it is a sudden brief intensification of a slowly fluctuating state in nutrition," and imagines "the changes as brought about through what we are accustomed to designate as 'trophic' nerve fibres." He is now investigating the subject from this point of view.

Meanwhile we must conclude (1) that the ultimate nature of the reflex is still an unsolved problem; (2) that muscular, vasomotor, respiratory and secretory changes may be concomitant with emotional reactions, but that none of them is exclusively responsible for the psychogalvanic reflex; (3) that the probable cause is to be found in the biochemical changes in the skin, which are brought about by nervous excitation, but that there is no evidence to show what is the nature of these changes, or through what nervous channel this excitation takes place.

Clinical and Psychological Study of the Reflex.

The psychological problems involved in a study of the psychogalvanic reflex would seem to open up wide fields for inquiry, and yet on this side little systematic work has been done. Whatever may be the actual nature and causation of the phenomenon, it still remains for the psychologist to determine its significance, and also the conditions under which it varies, and to investigate the mental processes concerned in its excitation. The difficulty in interpreting the results of different observers is, firstly, that they are obtained under varying electrophysiological conditions, and are not directly comparable; and, secondly, that

the usual variations obtained between different individuals are not much greater than those obtained in the same individual at different times under different conditions of health; and, thirdly, that no standard measurement under constant electrophysiological conditions has yet been determined by any observer for this amount of variation in the same individual.

Waller [49] has worked out the periodic variations of conductance of the palm of the hand for one subject, and has found that they have a diurnal periodicity, the resistance being greater during the night than during the day. The question as to whether there is any constant relation between the strength of the psychogalvanic reflex and the apparent resistance of the skin as determined at the beginning of the experiment must remain open. Waller [49] thinks there is, but is not yet able to state it with complete numerical evidence. On the other hand, Ricksher and Jung [34] state that there is no relation between the two. If this question were settled in favour of Waller's assumption, then equally valuable evidence from a clinical point of view might be afforded by recording the apparent resistance of the skin, without any observation of the reflex itself. This would be a continuation of the original work of R. Vigoroux [44], who pointed out in 1879 that the resistance of the skin was considerably diminished in cases of exophthalmic goitre, and increased on the anæsthetic side in patients suffering from hysterical hemianæsthesia.

The observations which have been made from the clinical standpoint are mainly on the value of the reflex in the diagnoses of hysterical conditions, particularly hysterical anæsthesia, and also in the differentiation of the psychoses. Veraguth [43] has recorded cases of tabes, syringomyelia, &c., to show that, after painful stimuli in anæsthetic and analgesic skin areas of organic origin, there is no psychogalvanic reaction, when the stimuli are not appreciated subjectively by the patient as such. He has shown the importance of bandaging the patient's eyes and of giving no verbal indication of the stimulus during such experiments. If the patient could see or hear the approach of the stimulus, a normal psychogalvanic reaction was then given after stimuli in the anæsthetic and analgesic areas, due to the psychical effect of expectation. He also records one experiment with a cat, in which the reaction was abolished during ether narcosis, and in which, after dividing the spinal cord in the lumbar region, and allowing the cat to regain consciousness, a painful stimulus to the tail gave no reaction, whilst one to the front paw gave the usual reflex. He showed, on the

other hand, that stimuli in anæsthetic skin areas of functional origin always gave the usual reaction, and this has been the experience of all observers. I have also made use of the reflex for the purpose of diagnosing a functional amnesia from an organic one, and in one case in which the patient had a complete loss of memory for all his war experiences, I was able, by means of his galvanic reactions, to trace out roughly the history of the patient's movements during the war, and to determine the place where he was "blown up." In addition, I have used it as the only trustworthy test to decide whether a deafness is a true organic one or not; the amount of functional deafness in organic cases can be determined in this way. If the patient states that he does not hear the stimulus, but at the same time gives a reaction, then that amount of deafness is functional. It is impossible to state at present whether in these cases a negative reaction always indicates an organic deafness, but, by analogy with the cases of organic anæsthesia, it seems probable that this is the case.

Before studying the variation of the reflex in the psychoses, it is necessary to investigate the mental processes concerned in its excitation. So far it has been tacitly assumed that the reflex is simply an indication of an emotional reaction. Though it is easy to show that all emotional stimuli provoke galvanometric deflections, it is not so easy to say whether the deflections provoked by the psychical stimuli may not also be due to the effects of intellectual processes. Radecki [33] concludes that "the phenomena take place in the human organism exclusively as an expression of our emotions and affective states." Binswanger [4] has also expressed a definite opinion on this question to the effect that "among mental activities only the affective processes, in Bleuler's sense, have any influence. Intellectual work or simple sensory impression have no effect." On the other hand, it is the experience of most observers (Peterson and Jung) [28], Veraguth [43], Starch [40], Gregor and Gorn [14], that mental effort does produce a small reaction. Starch [40] concludes that "all the different types of mental processes produced by the various stimuli were accompanied without exception by galvanometric changes. Emotional processes and muscular activity produced the widest deflections, while habitual mental activity and the process of visual attention produced the least deflections. Quiet mental activity, even when involving considerable effort, produced small galvanometric changes. The degree of intensity of emotional experiences corresponds very closely with the amount of deflection." It is impossible to say how much affect accompanies an intellectual process

such as an arithmetical calculation, due to the effort of attention. Very often a strong affect is produced by the surprise at the question, or by the embarrassment and possible annoyance that the solution of the problem may be incorrect, and this can only be decided by introspection on the part of the subject. Even if a pure intellectual process does produce a reaction, it is very small, and is in no way comparable with the deflections produced by an affective process, so that for all practical purposes it may be considered that among mental activities only the affective processes have any influence in exciting the reflex. This is also confirmed by the fact that after repetition of the stimulus, thereby reducing its emotive effect, the reflex becomes correspondingly diminished.

The next question, which does not seem to have occupied the attention of any of the observers, is whether the deflections are not equally due to the attempt to suppress the emotion aroused, and to prevent it from operating. As Waller [49] puts it, "the more perfectly an examinee can control the visible signs of the emotion, the more violently is the galvanometer deflected through the palm of the hand by reason of his suppressed emotion." Waller only makes this statement in support of the fact that the palmar electrical sign is not under voluntary control, but it would seem to have a greater significance than he attaches to it. Waller [51] is of the opinion that the phenomenon is not a psychogalvanic reflex, and prefers not to use the prefix "psycho," because in his view the phenomenon has nothing to do with the conscious mind. He states "it is not a cortical phenomenon, it is a subcortical phenomenon." I have been led to think that there is a constant correlation between the visible signs of emotion as shown by muscular movement, which is under voluntary control, and the psychogalvanic reflex, which is not directly under voluntary control. The greater the visible signs of emotion, and the greater the muscular movement, the less is the response on the galvanometer. From this point of view a high psychogalvanic reflex would indicate not merely an emotive response in Waller's sense, but in addition an increased sensitiveness on the part of the subject which may be partly due to experimental conditions and also an increased power of control. Whereas a low reflex would indicate that the subject was of diminished sensibility or had little control over his emotions, it might also mean that the subject was of low emotivity, as Waller says, but it would not necessarily be so. In my view then the phenomenon is as much cortical as subcortical, and is not a true indication of the emotivity of the subject.

Not only has the psychogalvanic reflex been evoked by emotional reactions concomitant with mental causes known to consciousness, but also it has been shown by Morton Prince and Peterson [32], Peterson and Jung [28], Binswanger [4] and others, that stimulation of ideas in a state of repression in the unconscious ("complexes") will also produce a reaction. Morton Prince and Peterson carried out experiments on Sally Beauchamp in her different states of personality, and showed that a memory of an experience which could not be recalled to consciousness provoked the usual reactions; they also showed that the reaction was the same in hysterical anæsthesia as in the normal, and that objects in the peripheral field of vision not consciously perceived were recognized sub-consciously (with Prince "co-consciously"). Radecki [33], working with Abramowski, showed that reactions could still be obtained through the sub-conscious by giving stimuli whilst the attention of the subject was concentrated on some mental problem, when the subject was not able to recall the fact that such stimuli had been given. Peterson and Jung [28] and particularly Binswanger [4] have done elaborate experiments on "association" tests to show that the galvanic phenomenon, like reaction time and alteration of reproductions, may give evidence of the existence of an unconscious "complex." These reactions have been so marked that they have given it as their opinion that reactions are evoked particularly by "complexes," and they have used it extensively as a "complex" indicator. Binswanger points out that an existing affect or lasting concentration of the attention on matters irrelevant to the experiment inhibits the psychical elaboration of the stimulus, and that, therefore, under these conditions the stimulus does not provoke much emotive reaction or give a psychogalvanic reflex. Prolongation of reaction time without a corresponding increase of the galvanic reaction may occur when there are purely intellectual difficulties, such as indistinct perception of the stimulus word, but more frequently under the influence of perseveration. Increase of the psychogalvanic reflex without corresponding prolongation of the reaction time may occur for purely linguistic reasons, when a genuine emotional reaction takes place, but is masked if an easy reaction word is at hand, favouring a habit reaction or sound association. In the latter case the galvanic reaction is the only way of discovering the real significance of the stimulus word. This significance is often not known by the conscious mind and can then only be established with certainty by psycho-analysis. The psychogalvanic reaction is therefore a much more delicate and correct "complex" indicator than the "reaction time" test.

The influence of hypnosis on the reflex has been recorded by Moravcsik [23]. He quotes only one case, but this is a particularly interesting one, from the fact that the reflex was almost completely abolished as the result of suggestion. The reaction to a pinprick, after the suggestion that the hand was hypersensitive, corresponded to 35 mm. on his scale, and after the suggestion that his hand had become anæsthetic the reaction was only 5 mm. After the suggestion that the subject was deaf and blind, auditory and visual stimuli provoked no reactions at all. This result is difficult to explain, and is not in accord with our usual experience of functional anæsthesia and deafness in hysterical cases. Also it is not in accord with the experience of Morton Prince and Peterson [32], already quoted. I got different results in some experiments I did with Waller two years ago, which were not reported, but of which photographic records were taken. These subjects, three men and one girl, had been hypnotized by me previously to the experiments two or three times, and were able to go into the somnambulic state quite easily; one of the men had had three teeth extracted and an operation for whitlow, and the girl had had two teeth stopped, with the extraction of a live nerve from one of them, under hypnosis, without any visible signs of the perception of pain or conscious memory of the operation. Records were taken both in the normal and hypnotic states, and there were no appreciable modifications in the galvanic reactions, either to painful or auditory stimuli. Moravcsik does not record the normal reactions of his subject, and does not state whether the subject was previously acquainted with the experimental conditions of the reflex. It would, for instance, be easy for a subject who had become acquainted with the physiological processes causing a deflection, and who gave normally a small deflection, due to familiarity with the stimuli, to produce a big deflection by taking a deep inspiration. On the other hand, there are undoubtedly some persons who can influence the rate of their pulse, and possibly even the surface temperature of their body by suggestion. Moravcsik [23] has recorded one such case in which the pulse-rate was altered from 84 to 104 and the temperature from 37·3° C. to 38° C. Presumably such subjects would be able to influence indirectly the psychogalvanic reflex. That voluntary effort can in certain cases affect the reflex in the direction of diminution is claimed by Radecki [33], who thinks that this effect is due to the influence on respiration, and also to the fact that voluntary effort produces attention which interferes with the emotional reaction evoked. The most probable explanation would seem to be that the

attention necessitated by voluntary effort prevents the full psychical elaboration of the stimulus, and consequent production of emotion, and therefore the galvanic reaction is diminished.

The effects of different stimuli vary in different individuals. In some there is a greater reaction to actual sensory stimuli, such as pin-pricks and burns, than to stimuli involving the ideational processes, such as the expectation of a burn or the recalling of unpleasant memories. These differences appear to have some diagnostic value. It is difficult at present to compare quantitatively the reactions of different individuals, as the electrophysiological conditions are rarely comparable, but general conclusions can be drawn from the work of different observers. In respect to data concerning the latent period the conditions are more comparable. They can be recorded with accuracy in the case of sensory stimuli, but in the case of stimuli where associative elaboration takes place it is impossible to say exactly when the emotive effect begins. All observers are agreed that the latent period in healthy persons may vary from one to five seconds. Peterson and Jung [28] have recorded that the latent period is lengthened in cases of dementia præcox, with katatonia. Abbott and Wells [1], in an investigation on maniacal-depressive stupor, have reported that "the latent times to sensory stimuli observed under stupor are within normal limits," and that the "perceptive processes in conditions of maniacal-depressive stupor are but slightly lengthened from the normal. The retardation may lie in a lessened complexity of the associations formed, or in the slower and feebler conversion of these associations into their motor expressions.

An important question is as to whether there is any correspondence between intellectual development and the quality and quantity of the psychogalvanic reflex. There certainly appears to be some correlation between intellectual development and the reactions to the two different classes of stimuli. The greater the intellectual development the more pronounced are the reactions to ideational stimuli. The same condition exists in cases of anxiety neurosis; whereas in cases of conversion hysteria the reactions to sensory stimuli appear to prevail over those due to ideational stimuli, but there is not sufficient data forthcoming to decide this with any certainty. M. D. Waller [52], as a result of her investigations on seventy-three medical students, concludes that intellectual efficiency as judged by examination results is associated with higher nervous sensitiveness in the psycho-galvanic reflex. It is certainly my experience that the reactions in subjects of poor intellect, as evidenced by their low standard at school, are rarely so marked as in

my intellectual subjects, and I have attributed this to the fact that the intellectual subject has usually a much greater control over his emotions and has a wider range in the association of ideas. Claparède [5] has reported experiments on four idiots of the lowest grade, in whom no psycho-galvanic reflex of any kind was obtained even after painful stimuli, in spite of the fact that the stimuli were perceived and were responded to by marked muscular reactions.

Gregor and Gorn (14) disagree with Claparède, and quote one case in whom they obtained normal reactions to sensory stimuli.

The chief work on the psychoses has been done by Peterson and Jung [28], Ricksher and Jung [34], and Gregor and Gorn [14]. Peterson and Jung have reported experiments on eleven cases of dementia præcox. They observed nothing especially noteworthy in the hebephrenic type, but in the katatonic forms, especially the acute forms, they observed variations from the normal, both in the quality and quantity of the reflex, the chief observation being that there was almost no reaction at all in acute katatonic stupor. They consider that this result is due to the fact that "the entire psychic activity is bound up with the morbid complex." This explanation might be plausible if it were not for the fact that a similar result can be obtained in other forms of insanity, arteriopathic dementia for example, and according to Claparède in idiots, so that this result would seem to be more in accord with the auto-intoxication theory and deterioration of the cerebral cortex. Gregor and Gorn have reported similar results in dementia præcox. They found that the reactions became less as the disease advanced, but that the reactions reappeared as the patients began to come out of the stupor, and became greater as the patients began to improve. On the other hand, in transitory psychogenic stupors the reactions were normal.

The reactions were also small in cases of hebephrenia, and the reactions to pain stimuli were greater than those to other stimuli, whilst in hebephrenic maniacal excitement the reactions were practically absent. A similar picture was obtained in cases of general paralysis. In manic-depressive cases the reaction was reduced at the height of the depressive phase, but in all cases of mania there was a very marked reaction. Gregor and Gorn concluded that "the observation of the psychogalvanic reflex has a definite clinical value: (1) in judging the effects of impressions in stuporose and inaccessible individuals; (2) in distinguishing benign psychasthenic 'regression' from similar conditions in the course of progressive maladies; (3) the objective characterization of affective conditions as a criterion of affective sensibility; (4)

the prognosis of melancholia and katatonia; (5) distinguishing maniacal states due to paralytic or hebephrenic excitement; (6) the diagnosis of malingering."

In conclusion, it can only be said that a very good case has been made out for further investigation into the psychological study of the psychogalvanic reflex. The observations so far recorded are small and the opinions of the observers are often conflicting. But it would seem that we have in the psychogalvanic reflex a valuable objective sign, which may be able to help us in the elucidation of the many unsolved problems in psychological medicine, and which we cannot afford to disregard.

REFERENCES.

[1] ABBOTT, E. S. and WELLS, F. L. "Psychogalvanism in the Observation of Stuporose Conditions," *Psychol. Rev.*, 1919, vol. xxvi, p. 5.

[2] AEBLY, J. "Zur Analyse der physikal. Vorbedingungen des psychogalv. Reflexes mit exosomatischer Stromquelle," *Inaug. Diss.*, Zürich, 1910.

[3] ALBRECHT, O. "Exper. Unters. über die Grundlagen der sogenannten galv. Hautelektrizität," *Monatschr. f. Psychiat. u. Neurol.*, 1910, Bd. xxvii, S. 365, 439, 552.

[4] BINSWANGER, L. "Ueber das Verhalten des psychogalv. Phänomens beim Assoziationsexperiment," *Journ. f. Psychol. u. Neurol.*, 1908, Bd. x, S. 149; Bd. xi, S. 65, 133.

[5] CLAPARÈDE, E. "Sur le phénomène psycho-électrique," *Extrait des Arch. des Sc. physiques nat.*, 1911, t. xxxi, p. 379.

[6] DU BOIS REYMOND, E. "Untersuchungen, &c.," 1849, Bd. ii, S. 261, 276.

[7] FÉRÉ, CH. "Note sur les modifications de la résistance électrique sous l'influence des excitations sensorielles et des émotions," *Compt. rend. de la Soc. de Biologie*, 1888, p. 217.

[8] FÜRSTENAU, R. "Die Stellung der menschlichen Haut in der elektrischen Spannungsreihe," *Zentralb. f. Physiol.*, 1906, Bd. xx, S. 194.

[9] GILDEMEISTER, M. "Ueber die physikalisch-chemischen und physiol. Vorgänge im menschlichen Körper, auf denen der psychogalv. Reflex beruht," *Münch. med. Wochenschr.*, 1913, Bd. xliii, S. 2389.

[10] *Idem.* "Ueber die im tierischen Körper bei elektrischer Durchströmurg entstehenden Gegenkräfte," *Pflüger's Arch.*, 1913, Bd. cxlix, S. 389-401.

[11] *Idem.* "Der sogenannte psychogalvanische Reflex und seine physikalisch-chemische Deutung," *Pflüger's Arch.*, 1915, Bd. clxii, S. 489-506.

[12] *Idem.* "Über elektrischem Widerstand, Kapazität und Polarisation der Haut," *Pflüger's Arch.*, 1919, Bd. clxxvi, S. 84-105.

[13] GIRARD, P. "Sur les relations osmotiques des globules rouges avec leur milieu, &c.," *Compt. rend. de l'Acad. des sciences*, 1913, t. clvi, p. 1401.

[14] GREGOR, A. and GORN, W. "Zur psychopathologischen und klinischen Bedeutung des psychogalv. Phänomens," *Zeit. f. d. ges. Neurol. u. Psychiat.*, 1913, Bd. xvi, S. 1-104.

[15] GREGOR, A. and LOEWE, S. "Zur Kenntnis der physikal. Bedingungen des psychogalv. Reflexphänomens," *Zeit. f. d. ges. Neurol. u. Psychiat.*, 1912, Bd. xii, S. 411-446.

[16] HALLION, L. and COMTE, CH. "Sur les réflexes vasomoteurs bulbomédullaires dans quelques maladies nerveuses," *Arch. de Physiol.*, 1895, t. vii, p. 90.

[17] HERMANN, L. *Handbuch der Physiologie*, 1879, Bd. i, S. 224.

[18] HERMANN, L., and LUCHSINGER, B. "Ueber die Secretionsströme der Haut bei der Katze," *Pflüger's Arch.*, 1878, Bd. xvii, S. 310.

[19] KNAUER, A. "Kritische Bemerkungen zu O. Veraguth's Buch, 'Das psychogalvanische Reflexphänomen,'" *Journ. f. Psych. u. Neur.*, 1909, Bd. xxiii, S. 1-2.
[20] LEDUC, S. "Les ions et les médications ioniques," 1906, Paris.
[21] LEVA, J. "Ueber einige körperliche Begleiterscheinungen psychischer Vorgänge, mit besonderer Berücksichtigung des psychogalv. Reflexphänomens," *Münch. med-Wochenschr.*, 1913, Bd. xliii, S. 2386.
[22] MARKBREITER, R. "The Effect of Atropine on the Emotive Response" (in collaboration with A. D. Waller), *Proc. Roy. Soc.*, 1919, B, vol. xci, p. 40.
[23] MORAVCSIK, M. E. "Experimente über das psychogalv. Reflexphänomen," *Journ. f. Phys. u. Neur.*, 1912, Bd. xviii, S. 186-199.
[24] MÜLLER, E. K., cited by PETERSON [27] from *Compt. rend. de la Soc. helvétique des sciences nat.*, September, 1904.
[25] MÜLLER, H. "Exp. Beiträge zur physikal. Erklärung der Entstehung des psychogalv. Phänomens," *Inaug. Diss.*, Zürich, 1909.
[26] MÜLLER, V. J. "Zur Kenntnis der Leitungsbahnen des psychogalv. Reflexphänomens," *Monatschr. f. Psychiatrie*, 1913, Bd. xxxiii, S. 235.
[27] PETERSON, F. *Brit. Med. Journ.*, 1907, vol. ii, p. 804.
[28] PETERSON, F., and JUNG, C. G. "Psycho-physical Investigations with the Galvanometer and Pneumograph," *Brain*, 1907, vol. xxx, pp. 153-218.
[29] PETERSON, F., and SCRIPTURE, E. W. "Psycho-physical Investigations with the Galvanometer," *Journ. of Nervous and Mental Diseases*, 1909, vol. v, p. 426.
[30] PHILIPPSON, M., and MENZERATH, P. "Analyse physiologique du phénomène psychoélectrique," *Arch. Intern. de Physiol.*, 1914, t. xv, p. 1-30.
[31] PIÉRON, H. "Les variations physiogalvaniques comme phénomène d'expression des émotions," *Revue de Psychiatrie*, 1910, p. 486-506.
[32] PRINCE, M., and PETERSON, F. "Experimentelle Unters. über psychogalv. Reaktionen von mitbewussten (unterbewussten) Vorstellungen in einem Falle vielfacher Persönlichkeit," *Journ. f. Psychol. u. Neurol.*, 1908, Bd. xiii, S. 249-262.
[33] RADECKI, W. "Recherches expérimentales sur les phénomènes psychoélectriques," *Arch. de Psychologie*, 1911, t. xi, p. 209-295.
[34] RICKSHER and JUNG, C. G. "Investigations on the Galvanic Phenomenon," *Journ. of Abnormal Psychol.*, 1908, vol. ii, p. 5.
[35] SCHWARTZ, cited by Philippson and Menzerath [30], from *Zentralb. Physiologie*, 1913, Bd. xxvii, S. 734.
[36] SIDIS, B. "The Nature and Cause of the Galvanic Phenomenon," *Journ. of Abnormal Psychol.*, 1910, vol. v, p. 74.
[37] SIDIS, B. and KALMUS, H. T. "A Study of Galvanometric Deflections due to Psychophysiological Processes," *Psychol. Review*, 1908, vol. xv, p. 391, and 1909, vol. xvi, p. 1-35.
[38] SIDIS, B., and NELSON, L. "The Nature and Causation of the Galvanic Phenomenon," *Psychol. Review*, 1910, vol. xvii, pp. 98-146.
[39] SOMMER, R. "Zur Messung der motorischen Begleiterscheinungen psychischer Zustände," *Beiträge zur psychiatrischen Klinik*, 1902, Bd. i, S. 143.
[40] STARCH, D. "Mental Processes and Concomitant Galvanometric Changes," *Psychol. Review*, 1910, vol. xvii, p. 19.
[41] STICKER, G. "Ueber Versuche einer objectiven Darstellung von Sensibilitätsstorungen," *Wiener klinische Rundschau*, 1897, Bd. xxx, S. 497.
[42] TARCHANOFF, J. "Ueber die galvan. Erscheinungen," etc., *Pflüger's Arch.*, 1890, Bd. xlvi, S. 46.
[43] VERAGUTH, O. "Das psychogalvanische Reflexphänomen," Berlin, 1909, Verlag von S. Karger.
[44] VIGOROUX, R. "Sur le rôle de la résistance électrique des tissus dans l'électrodiagnostic," *Compt. rend. de la Soc. de Biologie*, 1879, t. xxxi, p. 336.
[45] WALLER, A. D. "Sur la mesure de l'émotivité nerveuse," *Extrait du Bull. de la Soc. Roy. des Sci. med. et nat. de Bruxelles*, 1919, t. vi, p. 2.

[46] Waller, A. D. "Calorimetry by Surface Thermometric and Hygrometric Data," *Journ. of Physiol.*, 1894, vol. xv, p. 25.

[47] *Idem.* "Demonstration of the Psychogalvanic Phenomenon," *Journ. of Physiol.*, 1918, vol. vi.

[48] *Idem.* "The Galvanometric Measurement of 'Emotive' Physiological Changes," *Proc. of the Roy. Soc.*, 1918, B, vol. 90, p. 215.

[49] *Idem.* "Periodic Variations of Conductance of the Palm of the Human Hand," *Proc. of the Roy. Soc.*, 1919, B, vol. 91, pp. 17-31.

[50] *Idem.* "The Influence of Drugs upon the Electrical Conductivity of the Palm of the Hand," *Proc. of the Roy. Soc.*, 1919, B, vol. 91, pp. 32-40.

[51] *Idem.* "Mensuration de l'émotion nerveuse chez l'homme," *Extrait du Bull. de l'Acad. Roy. de Méd. de Belg.*, 1919.

[52] Waller, M. D. "Emotive Response of Seventy-three Medical Students," *Lancet*, 1918, vol. i, p. 510.

[53] Weiss, cited in Guilleminot's "Electricity in Medicine," p. 202.

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Forms of Peripheral Neuritis among Troops serving with the Egyptian Expeditionary Force, 1915-19.[1]

By F. M. R. Walshe, M.D.

During the recent campaign in Egypt and Palestine undoubtedly the commonest organic nervous disease among the troops was multiple neuritis. It was also the only one that could definitely be said to be associated with the prevailing conditions of active service. Post-diphtheritic paralysis formed the bulk of the cases, and it is to these that my last remark applies.

During the whole period of my service from 1915 to 1919, I saw no case of multiple neuritis unquestionably due either to dysentery or to malaria, nor did any instance of acute febrile polyneuritis as it has been described by Gordon Holmes or Rose Bradford come under my observation. Finally, there was no beri-beri in Egypt or Palestine, apart from imported cases from Mesopotamia and a few in 1915 from Gallipoli.

I propose to confine my remarks this evening to a certain group of cases of multiple neuritis, and, since many of the symptoms of this malady are common to all its ætiological varieties, it will save you much wearisome and familiar detail if I single out for remark such noteworthy features of my cases as may be more or less new to you, and that may throw some light upon the origin of post-diphtheritic paralysis.

Of this, I saw over 160 cases. They fell into two groups: the first consisting of the ordinary type following faucial diphtheria; the second, and in some ways the more interesting group, associated with extra-faucial infections of superficial skin lesions and of wounds by the

[1] At a meeting of the Section, held January 8, 1920.

diphtheria bacillus. The opportunity of seeing so large a series was unique, for over sixty came under observation.

However, before referring to the characteristic clinical picture presented by these I should like to make some general observations on the order of appearance of the signs and symptoms of multiple neuritis. Having access to a large Isolation Hospital, I was able to observe continuously a number of cases of diphtheria until their discharge to Convalescent and Command Depots. Many were examined almost daily from the date of admission, and others from the appearance of palatal paralysis in the second and third weeks of their illness. Multiple neuritis developed in several of these while under observation, and contrary to expectation, I found that diminution and abolition of the knee- and ankle-jerks were by no means the initial signs of the malady. Subjectively, aching pains in the legs on exertion, painful cramps in the calves during the night, numbness of the feet and, objectively, tenderness of the calf and plantar muscles on pressure, with a remarkable facility of the tendon-jerks, were the earliest phenomena. In six cases I was satisfied that the knee- and ankle-jerks underwent a definite preliminary exaltation before they began to grow sluggish. In many cases the ankle-jerks became feeble while the knee-jerks were still brisk, and they always disappeared earlier than the knee-jerk.

The point is, perhaps, of minor clinical interest. Nevertheless, in the circumstances in which the observations were made there was a definite value attaching to them. For in dealing with convalescents from diphtheria, the eliciting of the knee-jerks was commonly regarded as giving a clean bill of health to the patient in this respect, and as being a sufficient reason for disregarding any subjective symptoms of which he might complain. But in my experience this test was by no means adequate to exclude an early polyneuritis, and thus many men were sent prematurely to duty only to be readmitted to hospital within a week or two with a fully developed polyneuritis.

To return to the question of post-diphtheritic paralysis: during the winter 1916-17 and onwards there was an epidemic of diphtheria in the field and throughout the lines of communication, which extended from Palestine to the Delta. Although of mild character, on the whole it was fairly heavy in incidence, and, associated with it, I saw numerous cases of the ordinary type of post-diphtheritic paralysis. I am not able to give even an approximate estimate of the total case incidence in this outbreak, nor of the proportion of cases of diphtheria in which nervous complications ensued, but I think that the latter was high, and I

ascribe this to two causes: first, the small dosage of antitoxin, a single dose of from 4,000 to 6,000 units being the rule; and secondly, the premature return of the patients to conditions of physical activity. Often within a month of an apparently mild attack of diphtheria, a man resumed route marching at a Command Depot, only to fall out with pains and weakness of the legs on his first march. In such a patient, one could watch the development of a full-blown polyneuritis, which disabled him for at least six months. In many of these men the knee-jerks were still obtainable on readmission for a short period. Further, many mild cases were, I am sure, never recognized as such but passed under various diagnoses—for example, tabes dorsalis, neurasthenia, and "D.A.H.," and thus any estimate of the total number was impossible.

Of the cases of post-diphtheritic paralysis following the ordinary faucial inceptions, there is little of particular interest to record, except possibly the large proportion in which the usual palatal and accommodation pareses were followed by a fully developed polyneuritis, probably for the reasons I have already given.

Early in 1917 cases of a mild type of multiple neuritis began to appear. They were not associated with any history of sore throat, nor with any other evidence of faucial diphtheria. The common ætiological factors seemed absent also, and the only feature common to the cases was the recent occurrence of a crop of "septic sores," except in one case, in which there had been a chronic suppuration in a flesh wound of the arm of some months' duration. The significance of this history was not recognized at once. And although the possibility of a secondary diphtheritic infection of the skin lesions was thought of, it was dismissed because the multiple neuritis was neither preceded nor accompanied by palatal or accommodation paralysis. In fact, the first ten or twelve cases that I saw had no cranial nerve symptoms whatever. In every instance their general condition was good, though there was in all of them a mild degree of tachycardia ranging from 80 to 100 when at rest.

However, some light was thrown on the question in the April of that year by a case which I have already recorded elsewhere, which made it appear highly probable that diphtheria lay at the bottom of all these obscure cases of multiple neuritis. The case was this: a medical officer attached to the same unit as myself performed a tracheotomy upon a fatal case of laryngeal diphtheria. He was not aware of having scratched himself at the time, but next morning he noticed a painful bleb upon the dorsum of the first phalanx of the right thumb. This

broke down leaving a sore raw surface. The ulcer rapidly spread over the surface of the thumb and ultimately reached the size of 1 in. by ½ in. It was covered by a greyish slough and exuded a scanty watery secretion. On the fourth day he began to feel ill and to shiver. There was a lymphangitis up the arm and the axillary glands were swollen and tender. On the sixth day the diphtheria bacillus was isolated in pure culture from the wound and he was admitted to hospital. During the second week of the illness, severe symptoms of toxæmia appeared: vomiting, cardiac irregularity and albuminuria, and persisted for about ten days. Improvement then set in, the wound began to heal, and in the seventh week he was discharged to convalescence. The knee-jerks were still brisk at this time. During the following two weeks he improved considerably, was able to walk uphill and to swim several hundred yards in the sea without undue fatigue, but from the seventh week onwards he had begun to notice that the thumb over and around the healed scar was very numb to touch and even to pressure. This impairment of sensation spread over the thumb and radial half of the hand, then invaded the rest of the hand and the forearm. Simultaneously his hand became very clumsy in fine movements, there was a constant feeling of pins and needles in the fingers and his handwriting became very untidy and difficult to read. His grasp was weak. About a week after his return to duty, that is, in the tenth week, he began to find that walking upstairs was very exhausting and required effort; his knees felt as though they might give way under him. Writing became quite impossible not only on account of the ataxy but because of painful cramps in the small hand and forearm muscles. There was no cranial nerve involvement. Twelve weeks after the original infection he was readmitted to hospital. On examination I found a well marked polyneuritis, with absent tendon-jerks, moderate weakness, tenderness of muscles and the characteristic sensory loss. But in addition to this, the condition of the right arm at once attracted attention. It was considerably weaker than its fellow. Its muscles were more flabby and the distal half of the limb was distinctly thinner. All fine movements of the hand and the movements of the proximal segments of the limb were ataxic. There was complete loss to touch over the hand and the distal third of the forearm and gross loss of other forms of cutaneous sensibility.

Here, then, was a case undoubtedly diphtheritic in origin, in which with the absence of palatal and accommodation paralysis, there were an initial local onset of nervous symptoms related to the site of the infective focus, and a multiple neuritis.

A short time after, a second and similar case was observed, and this is the last case I shall quote: The patient had received a flesh wound in the left lumbar region at the battle before Gaza in April, 1917. Although freely laid open early in the course of treatment and subsequently treated with every new and old-fashioned antiseptic, it had resisted healing. During August he was allowed up more freely, but as soon as he began to walk about he complained of an increasing weakness, of "pins and needles," and of cramp in his legs. A provisional diagnosis of syphilitic ulceration and early tabes dorsalis was made and the man was transferred to my care. From a small unhealed patch at the centre of the keloid scar, the diphtheria bacillus was isolated in pure culture, while clinical examination revealed a fully developed multiple neuritis. In this case there was neither local nor cranial nerve affection. I say there was no local palsy, but there was a large area of sensory loss surrounding the wound, which could not be accounted for by the local surgical condition.

During the summer of 1917, several pathologists were reporting the presence of "diphtheroid rods" in septic sores occurring in areas where faucial diphtheria was present, and at the same time numerous cases of polyneuritis following crops of septic sores came under my observation. I may say that by septic sore I refer to a condition in which small areas of superficial ulceration appeared upon the dorsum of the hand, the extensor surface of the forearm and on exposed parts of the leg round the knee. This condition was extremely prevalent throughout the campaign, especially in mounted troops when on trek in the desert. They began to concern me because from the time that diphtheria became prevalent these skin lesions were undoubtedly associated with a considerable amount of multiple neuritis among the troops. Unfortunately, in the majority of cases the primary lesions were all healed by the time the cases came to me with nervous symptoms, but in addition to the two cases already described I ultimately saw nine cases in which the cautious pathologist was able to isolate "a diphtheroid organism morphologically indistinguishable from the Klebs-Loeffler bacillus."

In all, I examined over sixty such cases of polyneuritis, and in addition to their close association as to time and place with an outbreak of diphtheria and of diphtheritic paralysis, and to the finding of the organism in several of them, certain clinical signs that they showed convinced me that they were true post-diphtheritic paralyses following cutaneous infections. Some new features were seen later in the series

that the first cases had not shown, and an analysis of the whole number, made when I had observed thirty and confirmed by a second thirty seen later, revealed that the complete symptom complex consisted of three elements :—

(*a*) An initial local paresis related anatomically to the site of the infective focus.

(*b*) A paralysis of accommodation, and

(*c*) Polyneuritis.

The local symptoms usually preceded by two or three weeks the visual and general symptoms. They occurred in about a third only of the total number. The accommodation paralysis was also present in but a third of the cases, while the polyneuritis was present in all. In the majority of instances multiple septic sores on the limbs preceded the onset of nervous symptoms, but in several the infective focus was single—a gun-shot wound or a boil—and it was in these that the local paralysis was most commonly seen. In the first thirty cases, eight had a single primary focus, and in six of these there was an initial local onset. In none of the number was palatal paralysis observed, and in none were diphtheria bacilli found in the throat.

A brief consideration will show that these three elements are also found in post-diphtheritic paralysis as it follows the ordinary faucial infections, for the palatal paralysis is a true local palsy ; the musculature of the soft palate is clearly closely related both in the matter of proximity and in the more important respect of innervation to the local lesion on the tonsils and fauces. This formula of three elements in the clinical picture of a fully developed diphtheritic paralysis may, therefore, be applied to any case, no matter where the infective focus may be. The question at once arises as to the origin of the nervous complications of diphtheria, and the path by which the toxin reaches the nervous system. The view commonly held is that diphtheritic paralysis is the result of a blood-borne toxæmia of the nervous system in which certain centres in the brain-stem are picked out by the selective action of the toxin. And, since experimentally inoculated animals do not show cranial nerve involvement as a part of their nervous symptom-complex, the further opinion has been expressed that in animals, on the other hand, the spinal nerve centres are specially attacked.

It seems, then, reasonable to say that the cases I have briefly described do not accord with this view, and, indeed, cannot be explained by it. For if the nervous lesion in diphtheria be the result of a selective blood-borne toxæmia, its clinical manifestations should

not vary according to the site of the infective focus, but should be constant and unvarying, except in intensity. It appears to me that the following view best accounts for the presence of the three clinical elements I have described; it is of course an hypothesis that as yet lacks both experimental and pathological confirmation; it is as follows: That the initial local palsy is the result of the passage of the toxin from the infective focus to the central nervous system, brain-stem or spinal cord, along the perineural lymph channels of the peripheral nerves innervating the site of the local lesion. This is the process so extensively investigated by Orr and Rows and named by them "lymphogenous toxi-infection of the nervous system." The suggestion is therefore merely an extended application of a known pathological process. This is not to say that the symptoms are caused by an ascending neuritis, for we know from Orr and Rows that the nerve, as far as its nervous elements are concerned, is commonly intact, and that the lesion is central and is situated in that segment of the central nervous system from which the nerve arises. We may thus explain equally well the initial palatal paralysis of a case of faucial diphtheria, or the initial sphincter disturbances of a case of diphtheritic infection of an ulcer in the perineum.

Accommodation paralysis and polyneuritis are common to all types. For the former, therefore, we may still invoke the hypothesis of selective action, while the latter may be regarded as the expression of the general toxæmia as it affects the nervous system, while in both instances the toxin probably reaches the nervous system by the blood-stream.

It is of interest to determine whether the initial local symptoms indicate a peripheral or a central lesion of the nervous system, and it does not appear to me that the palatal symptoms of the ordinary type throw much light on this point. In the extra-faucial cases it is equally difficult to decide because the presence of a multiple neuritis masks any distinctive characters the initial palsy may possess. However, I was so fortunate as to see one case in which this was in a region usually unaffected in multiple neuritis. The case was one of ulceration in the perineum, arising as a septic sore (and later, presumably, secondarily infected by the diphtheria bacillus), which was followed by a local, an accommodation and a generalized nervous symptomatology. The local symptoms were sensory loss over the territory of the lower four sacral posterior roots on both sides and definite loss of control over the bladder, which, I think, can only be explained by supposing that the lesion was central and not merely an ascending neuritic process in the afferent nerves of the region.

I also saw a case—and one has been recorded by Oppenheim—of a streptococcal infection of the tonsils and fauces in which palatal paralysis and a multiple neuritis supervened. In both repeated search failed to demonstrate the presence of the diphtheria bacillus. Oppenheim remarks of his case that this clinical picture cannot be regarded as absolute proof of a diphtheritic infection, and I would suggest that these cases tend to confirm the view of the origin of the initial local phenomena that I have put forward. Several recent case reports in German periodicals of extrafaucial diphtheria and a resulting nervous involvement also bear me out in this respect.

In considering post-diphtheritic paralysis from the point of view I have put forward this evening, one cannot escape the analogy it presents to tetanus. Meyer and Ransom have divided the symptoms of this into three groups.

(*a*) *Local tetanus*, in which, after prophylactic inoculations of tetanus antitoxin, the first symptoms appear in the immediate vicinity of the infective focus.

(*b*) *Specific tetanus*, in which, irrespective of the site of the infected focus, there is spasm of certain muscles—namely, trismus; and

(*c*) *Generalized tetanus*.—If the amount of toxin be considerable a generalized tetanus is superadded to the two preceding forms.

After intravenous injections of tetanus toxin into animals, local tetanus is not seen; similarly, after intravenous injection of diphtheria toxin into experimental animals, the paralysis is generalized and presents no local phenomena. We may, therefore, compare the local, specific and generalized forms of tetanus with the palatal, ocular and generalized nervous symptoms of faucial diphtheria—the palatal palsy being the local element, the accommodation affection the specific element, while the polyneuritis corresponds to generalized tetanus.

In conclusion, I must ask your indulgence for the somewhat broad manner in which I have dealt with my subject. There are many gaps, I know, on the theoretical side of my paper. I have merely tried to describe a somewhat unusual type of peripheral neuritis, and to indicate briefly the theoretical considerations to which its study gave rise, and its bearing on the pathogenesis of post-diphtheritic paralysis.

I know also that this is by no means a comprehensive review of the various forms of peripheral nerve disease that one saw in Egypt. However, the rest of these were merely fortuitous in their occurrence and were in no way peculiar to that field of operations. I have thought

it best, therefore, not to attempt completeness, but only to deal with what seemed characteristic of the campaign in which I served.

DISCUSSION.

Dr. PHILIP MANSON-BAHR: I feel I am here under false pretences; what I am going to say as a sort of confirmation of Dr. Walshe's paper is the result, not of my own work, but that of my friend and late colleague, Dr. Craig, of Kendal. It was during the advance through the Sinai desert, and especially on the line of the Wady Guzze before Gaza, that "desert sores," or "septic sores," as they were called, first began to claim serious attention. Now from what we know this desert sore is probably synonymous with the "veldt sore" of South Africa and the "Barcoo rot" of Australia; at any rate it has something to do with desert conditions, and is quite distinct from the Oriental sore due to *Leishmania tropica*. Now it was noted by Craig that in the Egyptian Expeditionary Force the majority of units afflicted with these sores were mounted troops, or those associated with camels, and he concluded that as the clinical appearances of the lesions were so characteristic they must have a peculiar and specific cause. The sores affected those parts of the body exposed to injury—for instance, the hands, knees, and face. In mounted troops who rode in "shorts," as they did at that period, the sores were apt to form over the points of friction at the knee. However, in many cases they supervened *de novo* after no obvious sign of any injury. The onset of the sore is sudden and very painful, quite out of proportion to the size of the lesion; within a few hours a vesicle forms filled with serous, or it may be hæmorrhagic, fluid, collecting generally round a hair follicle. The vesicle soon bursts and leaves behind a raw area generally covered with a pearly-grey membrane and moist with exuding serum. The edges of the skin soon become undermined and the ulcer spreads in a peripheral direction and soon becomes punched out, chronic, and most intractable. The surrounding skin is livid and blue. Apart from the pain and disability they caused, the amount of debility, cardiac irregularity, and often anæmia, which existed in association with the sores, were sufficient to attract general attention and speculation as to the specific cause. Early in 1917 Dr. Charles Martin had reported the isolation of a "diphtheroid" bacillus from one of these sores, a fact which he did not then regard as significant, but rather he looked upon the lesions as being either streptococcal or staphylococcal in origin. This line Craig followed up, and in a short time was able to isolate diphtheroid bacilli, indistinguishable morphologically from the Klebs-Löffler bacillus, from 129 out of 197 of the sores he examined, that is, in 67·5 per cent. Making due allowance for the fact that many of his patients had been extensively treated with all manner of antiseptics, he reckoned that the percentage of successful operations should have been much higher. Almost pure cultures of Klebs-Löffler bacilli were obtained by Craig and myself from the serum which exuded from the vesicles on Löffler's glucose serum, but the longer

the ulcer had persisted, the more difficult it was to isolate the organism and the more numerous were staphylococci and streptococci. There was no doubt that they were typical Klebs-Löffler bacilli; they conformed to all the well-known tests, produced death in thirty-six hours when injected into guinea-pigs or quails, with hæmorrhagic peritonitis and lesions of the suprarenals; on the other hand, these animals could be efficiently protected by a simultaneous injection of antidiphtheritic serum. At the same time as these sores were so prevalent a large number of cases of faucial diphtheria were occurring, and most of our energies, as pathologists, were directed to cultivating the Klebs-Löffler bacillus in throat swabs by the thousand. But I must confess that we were more fortunate in obtaining a higher percentage of positive isolations from desert sores than from throat cultures. When General Allenby came out, he took, amongst other things, more than an active interest in these desert sores, as they were causing a great amount of disability; in some units as much as 40 per cent. of the men were unfit for hard labour and had their hands swathed in bandages. There was at this time, for all-sufficient reasons, a considerable amount of official reticence in acknowledging the importance of Craig's discovery. It was thought that the absence of fresh elements, and especially of beer, from the dietary had a mysterious influence upon these sores, so General Allenby caused all the beer in Egypt to be sent up the line. This had the effect of raising the spirits of the men, but it did not heal their sores. At this time we began to collect together a considerable amount of evidence of the association of these sores with paresis or neuritis of various parts of the body, as Dr. Walshe has just described. For instance, I remember the case of a corporal of the H.A.C. who was covered with these sores, who had neuritis of both legs, with lack of knee-jerks, hyperæsthesia of his calves, and great ataxia. He came down the line with a provisional diagnosis of tabes. Another I remember with paralysis of the palate following sores on the eyebrows and cheeks, and paresis and numbness of the arms, with multiple sores on the fingers and hands. Probably paralysis was much more common than was supposed, and in many cases it had been overlooked or attributed to presumably untreated faucial diphtheria. The effect of injection of 4,000 units of antidiphtheritic serum in the neighbourhood of these sores was almost magical; raw patches and ulcers which had persisted for months, nay, even years, after treatment with every form of antiseptic lotion and paste, healed up perfectly with sound firm skin within a week or ten days. I myself am absolutely convinced that this was so, though no success was obtained in getting this mode of treatment recognized officially or universally adopted. The scars which remained after healing are sufficiently characteristic, and the stigmata of "desert sores" will be evident for years; there is probably not a single man who served in the Australian Light Horse or Imperial Camel Corps who does not bear one or more. The hypothesis that these sores were a primary skin infection with the diphtheria bacillus led Craig to consider that they were in some way connected with desert transport, for they ceased to be a scourge directly we had moved forward into the more fertile and cultivated portions of Palestine. Suspicion fell on horse manure as a source of infection.

At that time the desert roads were laid upon a foundation of horse dung, which in this manner afforded a harder surface than sand for travel. Finally Craig succeeded in isolating a diphtheroid bacillus from this substance, but lack of opportunity prevented him from carrying his researches any further. From the experience I have had of this subject I should not be at all surprised to learn that a considerable number of cases of tropical polyneuritis, formerly diagnosed as beri-beri, are explicable on a similar basis. Finally, I would add that the inability of some pathologists to isolate the diphtheria bacillus from these ulcers in base hospitals in no way militates against Craig's work. By the time they reached the base these sores had been contaminated and invaded by all sorts of organisms, such as streptococci, which therefore cannot be considered the actual ætiological agent or the cause of the debility and neuritis associated with them. As an illustration I show a photograph of a Mesopotamian case recently under my care in which a sore suspected of being cutaneous leishmaniasis had persisted for two and a half years, and which instantly healed up after injection of antidiphtheritic serum.

Dr. FEILING: During my three years' experience in Mesopotamia I saw many cases of polyneuritis of various kinds in both British and Indian troops. On the whole it was not a very common disease there, at least amongst the British. I wish first to say a few words on the subject of beri-beri. While having the greatest admiration for the work done by many observers in attacking the problem of its ætiology from the viewpoint of a dietetic deficiency, I feel obliged to demur to this being the only cause or in all cases any essential factor at all. Beri-beri after all is at present only a clinical term and different infections or toxins can produce in the field of polyneuritis clinical pictures which are very difficult, if not impossible, to differentiate on clinical grounds alone. I would like to illustrate this by brief reference to two epidemics of polyneuritis with which I became acquainted in Mesopotamia. One affected a number of Chinese labourers. These men were all of a fairly superior class, skilled carpenters mostly, and were housed in two large huts. The cases were confined to men in *one* of these huts only. Their rations had been expressly designed to contain as many so-called "anti-beri-beri vitamines" as possible. They received good pay and were able to, and did, supplement their rations by extensive purchases in the local bazaars of such fresh food as fish, eggs, chickens and vegetables; I myself often saw them returning from market loaded with such purchases. Clinically the cases were typical beri-beri; many of them of the so-called "wet" or œdematous type. Another example of an epidemic of polyneuritis, which illustrated the difficulties of diagnosis and its consequent evils, was that in which a large number of cases of a severe form of polyneuritis occurred in a detachment of British troops occupying an isolated post in the desert. These cases were diagnosed as beri-beri, and many of them ultimately found their way to Bombay. There fuller investigations were undertaken with the result that arsenic was found in large quantities in the hairs of many of these cases.

Inquiries were instituted and it was eventually discovered that a barrel of white arsenic had by some means been included amongst the barrels of bleaching powder used for water disinfection, and that these unfortunate men had been steadily disinfecting their water supply for some time with white arsenic. With regard to *dysenteric* neuritis, I have seen a number of such cases in which a dysenteric infection, generally of the bacillary type, was followed by polyneuritis. Less frequently it occurred amongst amœbic cases. It has been suggested, though on no very clear grounds, that the emetine given therapeutically was the cause of the neuritis in the amœbic cases. I saw one rather remarkable example of this condition. The patient was a young officer, who was admitted to hospital for fever and shortly afterwards operated on and a large liver abscess evacuated; the *Entamœba histolytica* was found in the pus from the abscess. During convalescence he developed a very severe polyneuritis which in its clinical features was quite indistinguishable from beri-beri. *Typhus* fever has been the cause of a certain small number of cases. In one of such the signs suggested that the infection had fallen on the ganglion cells of the cervical enlargement of the cord rather than on the more distal part of the motor neurone, for the principal feature of the case was extreme atrophy of the muscles of the shoulder girdle, the whole picture closely resembling that produced by an acute anterior poliomyelitis of the upper extremities. The *influenza* epidemic of 1918 was responsible for many cases of polyneuritis, many of them of a very severe type. Some of these again bore a very close resemblance to the classical picture of beri-beri. I saw one case which showed the somewhat remarkable combination of extreme atrophy of the leg, with great œdema affecting the subcutaneous tissues of the trunk only; this case also showed diaphragmatic paralysis and a severe cardiac affection: in spite of the fact that malaria became a still further complication the patient eventually recovered completely. In conclusion, I would point out that in only too many of the cases the ætiology has been a matter of grave doubt or even pure speculation.

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EDITOR.

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BRAIN.

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APHASIA AND KINDRED DISORDERS OF SPEECH.[1]

BY HENRY HEAD, M.D., F.R.S.

IT is my pleasant duty to thank the Master and Council of St. John's College, Cambridge, for selecting me to deliver this lecture. It was founded close upon four hundred years ago by the famous grammarian

[1] The Linacre Lecture for 1920.

and humanist physician, Thomas Linacre, and I feel that I cannot bring a more suitable tribute to his memory than by attempting to penetrate the mysteries of aphasia and kindred disorders of speech.

This paper is a preliminary communication of conclusions to which I have been led mainly from the study of soldiers wounded in the war. I make no attempt here to correlate my results with those of other workers in the field; this I hope to do later in the Hughlings Jackson lecture, when I shall deal with the history of aphasia and kindred disorders. My object in the following pages is to put forward briefly a fresh conception of the clinical phenomena of loss of speech from the physiological rather than from the anatomical point of view.

Physicians first recognized that the acts of speaking, reading, and writing could be affected by a gross cerebral lesion at a time when many still believed in the "localization" of "human faculties" and "moral qualities" in various portions of the brain. At the beginning of the nineteenth century Gall had enunciated this theory, which had found wide acceptance. By a perverted process of reasoning from analogy, the under surface of the frontal lobes had been selected as the seat of the "sense and memory of words." This doctrine was hotly disputed, and by the middle of the century had begun to fall into disrepute. When, however, in 1861, Broca found that local disease of one half of the brain produced definite loss of speech, the theory of "localization" seemed to be confirmed, the more so since he placed the cause of the disorder in the third frontal convolution. The nature of the problem was, however, still obscured by the older idea that it was the "memory for words" or the "faculty of speech" which was situated in the affected parts of the brain.

But from the beginning (1868) Hughlings Jackson protested against the idea that there was a "faculty" of speech that could be destroyed by a cerebral lesion. These defects must be considered, he maintained, "on the psychical side as defects of mind, and on the physical side as defects of the nervous system." We have to consider speech on this wider basis, in order that we may be better able to see how speech is part of mind, and to get rid of the feeling that there is an abrupt and constant separation into mind and speech. "It is," he said in 1868, "the power of intellectual expression by 'movements' of any kind, which is impaired, those most special, as of speech, suffering most; those of simple sign-making, least or not at all." "The question" he adds, "is not, How is general mind damaged? but, What aspect of mind is damaged?'"

Most observers continued to cling to the terms "aphasia," "alexia," and "agraphia," and many cases were published which were supposed to be examples of these conditions in isolated form. But at the same time it was recognized that the acts of speaking, reading and writing, were based on more elementary processes; these were supposed to be "images" of movement, hearing and sight.

This assumption was purely gratuitous. In 1866 Jackson pointed out that, in some cases, images were entirely unchanged; in some, on the other hand, they were grossly disturbed, and to these he applied the special term "imperception."

But, with Wernicke's description of "Sensory Aphasia," the belief that certain instances of disordered speech were due to destruction of auditory or visual images seemed to become a demonstrable fact. Broca's aphasia was attributed to disturbance of the motor mechanism, whilst the other forms were said to be due to destruction of "auditory" or "visual word centres." The terms "motor aphasia," "word deafness," and "word blindness" became firmly established, although they were no more fitted to describe the actual loss of function than "aphasia," "alexia," and "agraphia." As time went on, it was evident that the actual disorders produced by a cerebral lesion did not correspond to any of these categories. Elaborate explanations were put forward, which tended more and more to be based on mechanical diagrams, founded neither on the anatomy nor on the known functions of the brain.

Finally, Marie made a bold attempt to bring theory into consonance with clinical facts; he put forward the view that "motor" aphasia was due to anarthria or disturbance of the higher articulatory mechanism, whilst the "sensory" form was in reality an expression of lowered intellectual capacity.

I hope elsewhere to enter more fully into the history of the various views on the nature of aphasia and kindred disorders. But this short statement must suffice to introduce the observations I am anxious to put forward in this paper.

Chapter I.—Material and Methods.

An inconstant response is one of the most striking results produced by a lesion of the cerebral cortex. During our studies in sensation, we found that a stimulus, exerting a constant physical force well above the normal threshold, was sometimes appreciated and at others evoked no reply. Moreover, a graduated increase of intensity did not of necessity

lead to an equivalent improvement in the answers given by the patient.

Bearing in mind this characteristic want of certainty in the reaction to measured stimuli, it seemed probable that disorders of speech due to cerebral injuries would reveal the same tendency. This is notoriously the case. It is not a sufficient test to hold up some object, and ask the patient to name it; at one time he may be able to do so, at another he fails completely. No conclusion can be drawn from one or two questions put in this way; his power of responding must be tested by a series of observations in which the same task recurs on two or more occasions.

Not only is it necessary to arrange the tests in sequence, but each set must be placed before the patient in several different ways. For example, six common objects are laid on the table in front of him, and he is asked to point to the one which corresponds to a duplicate placed in his hand out of sight. This is repeated for eighteen or twenty-four observations, so that the choice of any one object recurs three or four times in the course of the series. Then he indicates each one in turn, as it is named by the examiner, or makes his selection in answer to printed words set before him on a card. He next gives names to the various objects, one by one, and finally writes them down without saying anything aloud. The order in which the tests follow one another remains the same throughout; this alone makes it possible to draw any conclusion from the inconstant responses, which are so disconcerting, unless the answers are recorded in this manner. Moreover, this method enables us to learn how the patient responds to the same series of tests put before him in different ways.

Before we pass on to consider these tests more in detail, it is necessary to say something of the character of the patients on whom this research is mainly based. In civilian practice most of those who suffer from aphasia and kindred disorders of speech are men with arterial degeneration, and in many the blood tension is greatly increased. They are old, broken down, and their general intellectual capacity is diminished. Such patients are easily tired and are obviously unsuitable for sustained examination.

But the war brought under our care young men who were struck down in the full pride of health. Many of them were extremely intelligent, willing and anxious to be examined thoroughly. As their wounds healed they were encouraged and cheered by the obvious improvement in their condition. They were euphoric rather than

depressed, and in every way contrasted profoundly with the state of the aphasic met with in civilian practice.

There is still another difference between the results produced by gunshot injuries of the head and those vascular lesions which are usually responsible for disorders of speech in the old. The missile strikes the skull from without, and even if it penetrates the brain tends to cause the greatest damage on the surface. Many vascular lesions, on the other hand, destroy the substance of the brain where the fibres are diverging or converging on their path to or from the cortical centres; a small hæmorrhage may in consequence be followed by a profound and widespread disturbance of function. But structural changes produced by a local injury to the external surface of the skull, not only cause less severe and extensive manifestations of cerebral injury, but give greater opportunity for the appearance of loss of function in dissociated forms.

Moreover, with gunshot wounds of the head, the symptoms tend to clear up to a considerable extent, however severe may have been the effect produced by the initial impact of the bullet, provided there are no secondary complications. Some aspects of the disordered function recover more rapidly than others; then it is that the various cerebral activities associated with speech, reading and writing become revealed in isolated forms. As recovery proceeds, some one of them may remain permanently defective, or in the end the patient may recover his powers to such an extent that he no longer fails to carry out the rough and simple tests which can be employed in clinical research.

§ 1.—*Serial Tests.*

Before proceeding to describe in detail the actual tests used in this research, I wish to lay stress on certain general rules necessary for their success. The patient must be examined alone, in a quiet room, apart from all distracting sights and sounds. It is of fundamental importance to record not only what he says or does but also every remark or question of the observer. As soon as it is certain that the patient understands the task he is asked to perform, each series of tests must be carried out in silence; should this rule be broken, both sides of the conversation must be recorded. It is particularly important to write down at the moment any statement which throws light on the ideas or feelings of the patient with regard to the test, or to the difficulties he experiences in carrying it out. If it consists in executing some choice

to verbal command, the observer must say the words once only in the simplest and most direct manner. Should it be necessary to repeat the order at the request of the patient, the fact must be noted, so that we may learn in how far his subsequent conduct is influenced by the repetition. Between any two series of tests, it is well to permit the patient to rest or to talk freely; but as soon as a fresh set of observations has been started, all conversation should be confined to the task in hand, and every word spoken on either side must be recorded.

It is extremely important to avoid all fatigue or loss of temper. Some patients, especially the older aphasics, become depressed or angry when they fail repeatedly to carry out tests which are childishly simple. The sequence of the various sets of observations must then be rearranged, so that the next series belongs to a group that can be carried out easily. It is remarkable how quickly this restores the patient's equanimity. In all work of this kind fatigue and disappointment must be avoided by every possible means, even if necessary by terminating the sitting.

For it must be remembered that we are here dealing with a general function, and not with an affection confined to one half of the body. There are no normal parts that can be used as an indication of the patient's general condition, as was the case when we were investigating the sensory reactions of the cortex. It is not a question of local loss of attention or fatigue; the functions which are disturbed form part of the general activities of the mind.

This opens up another difficulty which was not present in our previous researches. Much in the character of the patient's answers depends on his previous aptitudes, which are entirely unknown. When testing sensation, this was of little importance, because every observation made on affected areas of the body could be compared with the response from equivalent normal parts. Since it is impossible to discover how the patient would have responded before he became aphasic, I have depended mainly on the reactions of young men wounded in the war; for, especially in the case of officers, it was possible to estimate with considerable accuracy the extent of their education, and the ability with which they had carried out the more exacting of their military duties. At the same time the profession or occupation exercised before the war frequently showed that they must have possessed faculties which were subsequently found to be grossly affected. Thus, in one case, an accountant could no longer carry out simple arithmetical operations, and a bank clerk had lost the power of adding up a column of figures with certainty.

The tests I am about to describe in detail vary greatly in the difficulty they present to persons of normal understanding. Most of them, such as naming common objects or colours, the man, cat and dog test, and the coin-bowl test, are childishly simple, and can be carried out perfectly by the stupidest individual. But many normal men are liable to make mistakes in the hand, eye, and ear test, when attempting to imitate the movements of an observer sitting opposite to him. This difficulty seems, however, to be lessened after the instruction in signalling, through which all our young officers were compelled to pass before obtaining a commission.

All the tests in any one group are not of the same order of severity. Some can be carried out with greater ease and rapidity even by normal persons, and these differences may become greatly exaggerated in pathological cases. This is particularly evident when the command requires choice between two or more courses of action. Moreover, words conveying an order always set a harder task than those which indicate an object. Told to put out his tongue, the patient may point to it, but be unable to protrude it; and yet he can do so perfectly to lick his lips.

Throughout these observations it is important to record the rapidity as well as the nature of the response. Frequently the number of erroneous answers is sufficient to show that the power to carry out some particular series of tests is defective. But occasionally every actual reply may be correct, and yet it is evident from the patient's statements and demeanour that he has difficulty in carrying out the task he has been set. A bare record of the number of times he has been successful in his choice would not reveal this defect. In many cases, therefore, it is necessary to time the interval between question and answer with a stop-watch. Speaking roughly, it will be found that the majority of rapid replies are correct, and any task performed with ease is carried out quickly.

(*a*) *Naming and recognition of common objects.*—Six objects of daily use,[1] such as a pencil, a key, a penny, a match-box, a pair of scissors and a knife, are laid on the table. Before starting the observations it is well to record the actual position in which they lie to one another from the patient's point of view. They are then screened from his sight, and a duplicate of one of them is

[1] Sometimes, when my patient was highly educated, I have replaced these articles of daily use by solid geometrical figures, having first ascertained that he was familiar with their proper names.

shown to him; the screen is rapidly withdrawn, and he is asked to point to the object on the table which corresponds to the one he has just seen. From eighteen to twenty-four observations are carried out in this manner, and patient and observer remain completely silent throughout. The power to carry out this form of the test is not affected by any of the disorders of function which are the subject of this research; but it is well to begin with a task that can be performed without difficulty by the patient in order to encourage him, and at the same time to gauge his general powers of comprehension.

Then he is asked to point to an object on the table which corresponds to the name said aloud by the observer. This series is carried out in exactly the same order as the first and all subsequent sets of observations.

Next, he is given cards each of which bears in printed characters the name of one of the objects in front of him, and he is asked to make his choice.

Both these forms of the test may give him considerable difficulty; and I am therefore accustomed to follow them up by placing a duplicate in the normal hand, out of sight, and asking the patient to choose the corresponding object from amongst those on the table. In all cases of uncomplicated disorders of speech of cerebral origin, this can be carried out perfectly.

He is then asked to write down in turn the name of each object indicated by the observer. This must be carried out in silence; but it is impossible to prevent many patients from moving their lips during the act of writing. After carrying out this test, it is often useful to make the patient name the objects aloud, and then write down what he has said. Occasionally he is asked to copy in cursive handwriting the printed name on the cards, or to repeat each name as it is said by the observer. These two tests are inserted when there is doubt as to the patient's power of repetition and copying.

Let us take as an example the answers to these tests given by No. 2, seven months after the injury to his head. (Table I, p. 95.)

Throughout the first series he never hesitated for a moment, but was prepared to indicate the correct object on the table, even before the screen was removed which hid them from his sight.

With the second series he was much slower. At first his lips moved, forming the word said by me, or he actually whispered or uttered it aloud. He said, "I can't remember what they are unless I think. If it wasn't here on the table I couldn't tell you."

TABLE I.

	(1) Pointing to an object on the table which corresponds to one shown to him	(2) Pointing to an object named verbally by the observer	(3) Pointing to an object named in print	(4) Duplicate placed in his left hand out of sight	(5) Naming an object indicated	(6) Writing the name of an object indicated
Knife ..	Correct and very quick	Correct (whispered "knife")	Correct ; silent	Correct and very quick	Correct..	Wrote "Jan"
Key ..	,,	Correct	,, ,, ...	,,	Placed his hand into his pocket, pulled out keys. Said "No, I can't tell you that"	(Gave up)
Penny ..	,,	,, (repeated "penny")	,, ,, ...	,,	Correct..	Wrote "panin"
Matches ..	,,	Correct (repeated "matches")	,, ,, ...	,,	"That's match, mat, mats, match"	Wrote "machin"
Scissors ..	,,	Hesitated, moved lips; then correct	,, ,, ...	,,	"That knap, ker, kur-te . . . No, I can't tell you that"	Wrote "nare"
Pencil ..	,,	Correct, quick and silent	,, ,, ...	,,	"Pentl, permitch ; not quite it. Pĕ night. No, I can't"	Wrote "knelers"
Key ..	,,	Correct ; slow ; silent	,, ,, ...	,,	"That's mer, may".. ..	Wrote "no, karest"
Scissors ..	,,	Hesitated ; then correct ; silent	,, ,, ...	,,	"No idea. I know exactly what it is, but I can't tell you"	(Gave up)
Matches ..	,,	Correct ; quick ; silent	,, ,, ...	,,	"That's match"	Wrote "no merest"
Knife ..	,,	Hesitated ; finally correct ; silent	,, ,, slow ..	,,	"Nĕ-ife, Ker-nife"	Wrote "knerreg"
Penny ..	,,	Correct : slow ; silent	,, ,, very quick	,,	Correct	Wrote "pe. penner"
Matches ..	,,	Correct ; slow ; silent	,, ,, ...	,,	"That's . . . match" ..	Wrote "mar, matare"
Scissors ..	,,	Correct ; quick ; silent	,, ,, ...	,,	"That's . . . No"	(Gave up)
Pencil ..	,,	Very slow ; finally correct ; silent	,, ,, very slow	,,	"It's a p. something . . . No"	(Gave up)
Penny ..	,,	Correct ; quick ; silent	,, ,, ...	,,	Correct..	Wrote "penne"
Knife ..	,,	Very slow ; finally correct ; silent	,, ,, ...	,,	"Kĕ-nife. I always remember that . . . It's canif in French"	Wrote "kerrert"
Key ..	,,	Correct ; quick ; silent	,, ,, ...	,,	Took his keys out of his pocket and said "Mat, latch"	Wrote "wer, karet"
Matches ..	,,	Correct ; quick ; silent	,, ,, quick ..	,,	Correct..	Wrote "marest. maters"

When he pointed to an object named in print, the card was held in front of him all the time until he made his choice. His finger hovered in the air, whilst he looked backwards and forwards at the card, until at last he dropped his finger onto the correct object. Except where he made his choice rapidly he reinforced his memory by frequently consulting the printed name on the card in front of him.

When a duplicate was placed in his hand out of sight, he never had a moment's doubt, but indicated the object with great rapidity. Moreover, he remained silent and his lips did not move.

Asked to name an object to which I had pointed, he showed great hesitation and difficulty. His lips moved and he frequently made several attempts in vain to find the word. Occasionally, however, he enunciated it at once, and without preface, but sometimes a correct answer was preceded by "That's . . ."

Throughout his attempts to write the name of an object shown him, he said nothing aloud; but it was obvious from the movements of his lips that he was attempting to find the word with the aid of silent articulatory movements. His power of naming was, however, so bad that it gave him little help in writing the name of the object.

(*b*) *Naming and recognition of colours.*—This test is carried out in the same manner as the one I have just described, except that coloured silks are substituted for the objects of common use. Eight strips of different colours are laid in a row and hidden from the patient by a sheet of cardboard; he is then shown an exact duplicate of one of the pieces on the table and, when the screen is removed, is asked to indicate the one which corresponds with it. Should there be hemianopia or any defect of the visual field, it is important to make sure that the whole gamut of colours is visible to the patient from the position in which he is sitting. Provided this source of error is avoided, the test in this form could be carried out correctly in all the cases of unilateral lesion of the brain comprised in this research.

Then the patient is asked to indicate the colour named orally or in print. Next he says the name of each one in turn or attempts to write it down silently. Lastly, he reads the name of each colour aloud, as it stands on a printed card.

The following example (Table II, p. 97), taken from No. 2, shows many of the most characteristic errors. At first sight it might seem as if he were colour-blind; but this is excluded by the rapidity and correctness with which he matched colours in the first series of observations. The defect was one of names and the gross errors were due to want of

TABLE II.

	(1) Pointing to similar colour to that shown	(2) Pointing to colour named by the observer	(3) Pointing to colour named in print	(4) Naming colour shown	(5) Writing name of colour indicated	(6) Printed name read aloud
Black ..	Correct and rapid	Pointed to white saying "Black"	Chose white ..	"Green—red; no, not red. I can only call it dead."	Wrote "Red" ..	"Red"
Red ..	,,	Correct, whispered "Red"	Chose red very slowly	"Ber-lu. I know what it is. It's what the Staff . . . the same colour I had here. I think it's red"	Wrote "Bell" ..	"Green. No, that's red, blue. No, it isn't blue, it's red"
Blue ..	,,	Correct, whispered "Ber-lu"	Chose violet ..	"Ber-lu"	No response ..	"Green"
Green ..	,,	Correct, whispered "Green"	Chose white ..	Whispered "Green." Compared it with blue band on his arm and rejected it. "I don't know this one. . . . Ber-lu"	Wrote "Gren"	"Green"
Orange ..	,,	Chose yellow, said "O-age." Then chose orange	Chose orange ..	"It's like Pil . . . I think that's red"	Wrote "Read"	"Red"
White ..	,,	Chose yellow silently ..	Chose yellow ..	"That's green"	Wrote "Gernd"	"Red, that's red"
Violet ..	,,	Correct; silent	Chose violet ..	"Ber-lu, ber-loor. . . It's more like mauve"	Wrote "Moved"	"Blue, green; no, mauve"
Yellow ..	,,	"I have no idea. Yellow. I don't know." Finally chose orange	Chose white ..	"That's red"	Wrote "Grena"	"Red"
Red ..	,,	Said "Pred" and chose red	Chose red ..	"What the Staff is again. I can't remember what it is"	Wrote "Blead"	"Red"
White ..	,,	Said "White" and chose white	Chose white ..	"Green"	Wrote "Red" ..	"Green"
Yellow ..	,,	Correct; silent	Chose violet ..	"That is . . . I know what I am trying to remember . . . Kark-too, tark-loon, kar-ki . . that's right"	Wrote "Run" ..	"Red"
Blue ..	,,	Said "Ber-lu; I think it's this one." Very slowly chose blue	Looked at the blue band on his arm, and after great delay chose blue	"Ber-lu"	Wrote "Movy"	"Ber-lu"
Green ..	,,	Said "Dreen, green." Chose white	Chose black ..	Touched his arm, shook his head and said "Mauve. It's not quite mauve, but it's rather like, I think"	Wrote "Gernat"	"Red"
Black ..	,,	Said "Black" and chose correctly	Chose blue ..	"That's red"	Wrote "Mat" ..	"I know this is an ordinary one. Green. No; red; no"
Orange ..	,,	Said "Or-ridge." Choseyellow	Chose orange ..	"I think it's pink"	Wrote "Grend"	"Mauve"
Violet ..	,,	Said "Violet." Very slowly chose violet	Chose violet ..	"Mauve"	Wrote "Moved"	"This one is mauve"

nominal recognition ; even the printed names, when read aloud, showed the same striking defects as the choice of the colour to oral command.

(c) *The man, the cat, and the dog test.*—This test is designed to investigate the power of reading and writing in its simplest and most elementary form. Every word employed comprises three letters only ; the patient is, therefore, precluded from guessing at the constituents of the phrase by their length, a fallacy which is otherwise liable to vitiate all observations of this class. But, on the other hand, the task set is childishly easy, and the defect in speech must be severe, before it shows traces on so simple a test.

First, the patient is made to read aloud the different combinations from printed cards. Then he is shown pictures of a man, a cat, and a dog in pairs, corresponding to those he previously read aloud. Next, he is asked to write the phrases from pictures or from dictation. Finally, he is made to repeat the words said by the observer, to read what he has written, and to copy from the printed card.

The commonest form of error revealed by this test, as shown on the following table, is a tendency to substitute one name for another. Occasionally this may go so far that the patient inserts "woman" into his answer, oblivious of the fact that no word of this length occurs in the series.

I have chosen for reproduction a set of observations where the six combinations were put once only; it is, however, advisable to repeat them once or more, varying the order each time. (Table III, p. 99.)

(d) *The clock tests.*—Two clock faces are prepared, about twelve centimetres in diameter, fitted with metallic hands, which are adjustable. On each, the figures of the hours are clearly marked in Arabic numerals. One of these clocks is given to the patient, and he is asked to place the hands in an exactly similar position to that of the other one, set by the observer. This direct imitation can be carried out correctly, unless the power of comprehending the nature of the task is considerably diminished.

Next, the patient is told to set the clock to oral and then to printed commands, and in each instance the sequence of tests is the same. The hands of the clock are now moved by the observer into various positions and the patient tells the time aloud, or without speaking writes them on paper.

Most of the characteristic errors discovered by this set of tests can be seen on the following table, compiled from observations on

TABLE III.

	(1) Reading aloud	(2) Reading from pictures	(3) Writing from pictures	(4) Writing from dictation	(5) Repetition	(6) Reading what he has written	(7) Copying
The dog and the cat	Correct; quick	Correct	Correct	Correct	Correct	Correct	Correct
The man and the dog	"The man and the . . . dog" (slow and hesitating)	,,	,, ...	,, ...	,,	,,	,,
The cat and the man	Correct; quick	,,	,, ...	"The cat and the dog"	,,	,,	,,
The cat and the dog	"The cat . . . the cat and the . . . dog" (slow and hesitating)	,,	,, ...	"The cat and the man"	,,	,,	,,
The dog and the man	Correct; quick	,,	"The cat and the man"	Correct	,,	,,	,,
The man and the cat	Correct; quick	,,	Correct	"The man and the dog"	,,	,,	,,

TABLE IV.

	(1) Direct imitation	(2) Clock set to oral command	(3) Clock set to printed command	(4) Telling the time shown on a clock set by the observer	(5) Writing the time shown on a clock set by the observer
5 minutes to 2 ..	Correct..	Set 2.10	Set both hands at 2 ..	Correct	Wrote nothing. Said "I seem to be getting confused between the hands".
Half-past 1 ..	,, ..	Set 1.10	Set both hands at 1 .. "I seem to think there is something wrong"	,, ...	Correct
5 minutes past 8..	,, ..	Both hands set at 8. "I've forgotten"	Set both hands at 8 ..	,, ...	"Ten minutes past eight"
20 minutes to 4 ..	,, ..	Both hands set at 4. Then set 4.25	Long hand at 4; short hand at 7	"25 minutes to 4." ..	Gave up
10 minutes past 7	,, ..	Long hand at 7; short hand at 5	Correct..	"10 minutes past 8 . . . 7"	Correct
20 minutes to 6 ..	,, ..	Set 6.20	Set 6.15	"25 to 6. . . I seem to get in a maze, when I go back to it again"	"Ten minutes past seven"
10 minutes to 1 ..	,, ..	Set 1.10	Set both hands at 2 ..	"5 to 8. No, that's 10"..	Correct
A quarter to 9 ..	,, ..	Correct	Correct..	Correct	"Quarter to eight"
20 minutes past 11	,, ..	Long hand at 11; short hand at 10	Long hand at 11, "I've no certain opinion. It will be a case of tossing up"	"10 minutes past 11. . . I get confused between the long hand and hour hand"	"Ten minutes past four"
25 minutes to 3 ..	,, ..	Shook his head. Set long hand at 3. Then gave up	Set 3.35	"Quarter past 7".. ..	"Half past seven"

No. 10. (Table IV, p. 100.) The patient mistook "to" and "past" the hour, or confused the two hands; thus he set the long hand at the hour and either placed the short hand in some erroneous position, or became confused and gave up the attempt. Except when he was imitating the position of the hands on the second clock set by the observer, he usually placed the short hand exactly on the number of the hour. For example, when asked to set "twenty minutes to four," he placed it opposite four, so that when the minute hand was brought to seven, the ordinary reading would appear to be 4.35. This inability to divide the space between the figures of the hours led to much difficulty in recording the position into which the hands had been placed, unless that assumed by each one was noted separately.

(*e*) *The coin-bowl test.*—Four bowls or saucers are set upon the table and in front of each is laid a penny. The patient is told to count them from left to right; he is then shown the nature of the task he has to perform, which consists in placing a coin into one of the bowls according to a series of numerical commands. First, the order is given orally, or by means of a printed card, which is read silently. Then the patient is asked to read it aloud, and to carry out the action demanded under the influence of words spoken by himself.

This is so simple a test that no normal person fails to carry it out correctly; but in some instances of aphasia and kindred disorders it may cause considerable difficulty, as seen by the example given on table. In this case both verbal and printed commands were poorly executed, although when asked to read the order aloud he did so correctly. (Table V, p. 102.)

It is sometimes useful to make two sets of observations with the printed cards under somewhat different conditions. First, the orders are given in numerals only, e.g., "1st into 3rd"; then the whole phrase is set out in full, as for example, "First penny into third bowl." In cases of aphasia the second form of this test usually presents greater difficulty to the patient, and may reveal defects that do not appear when the command is given in numbers only.

(*f*) *The hand, eye and ear tests.*—First of all the patient, seated opposite the observer, attempts to imitate a series of movements which consist in touching an eye or an ear with one or other hand. Before beginning these tests it is well to make sure that he knows his right hand from his left, and understands the nature of the task he has to perform. He is asked, therefore, to name each hand as it is raised by the observer, and then to lift the one which corresponds

TABLE V.

	(1) Oral command	(2) Printed command (not read aloud)	(3) Printed command read aloud	
			(a) Said	(b) Action performed
Second into third	Slow; correct	Correct	"Two . . . Three" ..	Correct
First into third	Hesitated; finally correct ..	Very slow: much hesitation; correct	"One . . . Three" ..	"
Second into first	Second coin . . . "I forget which one." Order repeated; quickly correct	First coin . . . Corrected to second coin. No further response	"Two . . . One" ..	First coin . . . into first bowl, saying "One, one"
Third into second	Third coin . . . "I forget which it was." Order repeated; correct	Second coin into third bowl ..	"Three . . Two" ..	Third coin . . . into third bowl
First into fourth	First coin . . . No further response. Order repeated; correct	First coin . . . After two minutes' hesitation, correct	"One . . . Four" ..	First coin . . . "Was it four?" pointing to fourth bowl. "I forget." Did nothing further
Fourth into third	Third coin into second bowl ..	Third coin into fourth bowl ..	"Four . . One . . Three"	Third coin into third bowl
Second into fourth	Correct	Correct	"Two . . . Four" ..	Correct
Fourth into first	Fourth coin . . . "I forget which one." Order repeated; correct	Correct	"Four . . . One" ..	First coin. "Four" . . . into fourth bowl
Third into first	Correct	Third coin . . . "I forget which one I've got"	"Three . . One . . Three"	Correct
First into second	Second coin to first bowl; quickly	Correct	"One . . . Two . . . Two"	Correct. "Was that it? I don't know"
Third into fourth	Extremely slow but correct ..	Third coin . . . "I forget which one to put in. It's one of these," pointing to third and fourth bowl	"Three . . . Four" ..	Third coin into third bowl. . . . Removed it and replaced it in third bowl
Fourth into second	"I forget which it was." Order repeated: fourth coin into first bowl	Correct	"Four . . . Two" ..	Correct

to it, bearing in mind the face to face position. When we are certain from these preliminaries that he appreciates the action demanded of him, the observations are begun.

Some normal persons find difficulty in performing these movements correctly over a series of from sixteen to twenty-four tests; many, however, can carry them out perfectly, especially if they are young and intelligent, and belong to the class from which so many of my war patients were drawn. There is a natural tendency to select the hand opposite to that used by the observer; this error is in most instances checked consciously. But none of the normal men I have examined failed to recognize that when the left hand was in contact with the right ear it had crossed the face; and yet this want of appreciation of crossed movement was one of the commonest pathological mistakes. Moreover, in certain cases, I have been able to watch a steady improvement in the records, resulting finally, after a considerable lapse of time, in a perfect series of answers.

Then the patient is placed in front of a large mirror, and is asked to imitate the reflected movements of the observer standing behind him. In all normal persons and in most of those suffering from disorders of speech, this can be carried out perfectly; it is an act of direct imitation uncomplicated by considerations of right and left, and not attended by the necessity for internal speech.

The next form of this test consists in handing the patient cards, each of which represents a human figure carrying out one of the desired movements. These drawings are simplified to the highest degree consistent with their significance, and a line is drawn down the centre to separate the right and left halves of the body. Most patients, when shown these pictures, make exactly the same kind of mistakes as when seated opposite the observer. But as soon as they are allowed to see the reflection in the glass, every movement may be executed rapidly and correctly.

Then the patient is made to carry out the same series of actions in response to oral and to printed commands. He is next asked to read aloud each order, and to execute it under the reinforcing influence of words said by himself. Finally, he is asked to write down in silence the movements made by the observer sitting opposite him. Occasionally, where the power of writing is in question, he is asked to copy the orders printed on the cards.

Table VI, p. 104, illustrating these tests, was obtained from observations on No. 4, a case of so-called "motor aphasia."

TABLE VI.

	(1) Imitation of movements made by the observer	(2) Imitation of movements of the observer seen in the glass	(3) Carrying out pictorial commands	(4) Pictorial commands seen in the glass	(5) Oral commands	(6) Printed commands (not read aloud)	(7) Reading aloud and executing printed commands	(8) Writing down movements made by the observer
Left hand to left eye	Correct	Correct	Right hand to right eye	Correct	Correct	Correct	Correct	"Left left eye"; correct
Right hand to right ear	Left hand; then right hand; correct	,,	Left hand to right ear	,,	,,	,,	,,	"Right hand right ear"; correct
Right hand to left eye	Correct	,,	Left hand to right ear	,,	,,	,,	,,	"Right hand left eye"; correct
Left hand to right eye	Left hand to left eye ..	,,	Right hand to left eye	,,	,,	,,	,,	"Left hand, right eye"; correct
Left hand to left ear	Correct	,,	Correct	,,	,,	,,	,,	"Left hand" ("I forget which it was")
Right hand to right eye	Correct	,,	Left hand to left eye	,,	,,	,,	,,	"Right hand right eye"; correct
Left hand to right ear	Left hand to left ear; "I have to think"	,,	Right hand to left ear	,,	,,	,,	,,	"Left hand right ear"; correct
Right hand to left ear	Right hand to right ear	,,	Left hand to right ear	,,	,,	,,	,,	"Right hand left ear"; correct
Left hand to left eye	Right hand; then correct	,,	Correct	,,	,,	,,	,,	"Left hand left eye"; correct
Right hand to right ear	Correct	,,	Left hand to left ear	,,	,,	,,	,,	"Right hand" ("I think it was the same side")
Right hand to left eye	Right hand to right eye	,,	Left hand to right eye	,,	,,	,,	,,	"Right hand left eye"; correct
Left hand to right eye	Right hand to right eye; then right hand to left eye; "I see the mistake"	,,	Right hand to left eye	,,	,,	,,	,,	"Left hand to right eye"; correct
Left hand to left ear	Correct	,,	Correct	,,	,,	,,	,,	Wrote nothing ("I've forgotten")
Right hand to right eye	Correct	,,	Correct	,,	,,	,,	,,	"Right hand right eye"; correct
Left hand to right ear	Correct	,,	Right hand to left ear	,,	,,	,,	,,	"Left hand right eye"
Right hand to left ear	Left hand to left ear ..	,,	Left hand to right ear	,,	,,	,,	,,	"Right hand left ear"; correct

This is the most difficult of all the serial tests, and the only one where the answers of a normal person may be at fault. But, if we bear this possibility in mind, it is capable of giving valuable information concerning the nature of the different defects of speech of cerebral origin.

§ 2.—*Further Tests employed in this Research.*

It is customary to test a patient, who suffers from one of these disorders of speech, with the alphabet and, if he shows any difficulty in naming the letters, he is given a pencil and told to write them in their proper order. But this is not sufficient to determine his disability; his capacity to carry out this test must be examined systematically. He is told to say aloud, to write and to read the alphabet; then he takes it down from dictation, copies it from printed capitals in cursive handwriting, and finally, if the formation of the sounds is defective, he is asked to repeat it letter by letter after the observer. Occasionally he is given the letters of the alphabet printed on separate cards, and is asked to arrange them in order.

A similar set of observations are carried out, using as a basis the days of the week or the months of the year.

Another valuable method of examination belonging to this order is to choose some paragraph in the newspaper of interest to the patient, and then to ask him to retail the information in different ways. For example, he is allowed to select an account of the latest boxing match, and asked to read it to himself silently; after the paper has been removed he narrates what he has gathered, and then writes down his own account of the fight. Then he is asked to write it from dictation, to copy the printed account in handwriting, and to repeat it verbally; phrase by phrase, after the observer. Finally, he attempts to read aloud what he has written, however defective it may be. The order of these operations is determined by the class of patient with whom we have to deal, and the nature of his defect. It is extremely important, however, that the task selected should be of interest to him, and it must not be long or difficult to remember; moreover, it should be strictly adapted to the extent of his education.

It is customary in these cases to place a picture before the patient, and to ask him to describe in spoken or written words what he sees. Sometimes a combination of this pictorial test with the comprehension of printed matter in the following way makes a useful method of

examination. A picture is chosen to which is attached a short printed description, so common in the daily press. At first the legend is covered up; the patient is asked to say what he sees in the picture, and the manner and nature of his observations are carefully recorded. Then he is allowed to see the printed description; after reading it silently to himself he writes down what it conveys to him, reinforced by the pictorial representation. He then reads it aloud and writes it to dictation. Finally, he is told to fix his attention on the picture, and to say what it conveys to him. An excellent example of this combined test is given on p. 139.

To be of any value arithmetical tests must be simple and carried out systematically in the order of their progressive difficulty. I am in the habit of beginning by asking the patient to add two simple numbers of three figures each, such as 235 and 462, which do not necessitate carrying over. This is followed by a couple which require one act of carrying over, and then by another where it is necessary to perform this operation twice.

From this he passes to the subtraction of one number of three figures from another, where no carrying over is required; the difficulties are then increased progressively in exactly the same manner as with addition. All these tests are absurdly simple to the normal man, provided he is not illiterate.

Great stress will be laid on the patient's ability to play games, such as chess, draughts, cards and billiards. "Jigsaw" and other puzzles may also give valuable indications of his aptitudes and disabilities.

His power of drawing must be tested in a more systematic manner than is usually the case. First, he is asked to draw from a simple model, such as a bottle or a vase. If he is fond of drawing and naturally intelligent, I sometimes put before him a glass spirit-lamp, because of the difficulties presented by the wick; for the parts above and below the collar must be shown as a continuous structure interrupted by the metallic neck of the lamp. This forms a useful problem in significance. Then both the model and drawing are removed, and he is asked to reproduce it from memory.

Another valuable test, which has been used by Marie and others, is to ask the patient to draw an elephant. Fortunately most of my patients were familiar with the appearance of these animals, and many had seen them in India during their military service. If possible, the patient should also be induced to draw images which come into his mind spontaneously, apart from any external command.

He is also asked to sketch roughly a ground plan of some room with which he is familiar, such as his ward in hospital. In some instances this is carried out with ease, whilst in others it is entirely impossible. But although the patient may be unable to put down on paper any of the significant objects in the room, his visual images are not of necessity defective. For if I draw an oblong in the centre of the paper to represent his bed, he may be able to indicate to me the position of the windows, doors, furniture, and other appurtenances of the ward with absolute correctness. He may know their position, but be unable to represent their relation to one another.

So far, all the tests I have described are new or, if commonly used, are employed by me in an unusual manner. They are not in any way complete, and are capable of profound improvement; but I have set them out in detail to show what methods have actually been employed in this research.

Chapter II.—Previous Explanations of Aphasia.

Gross injury confined to one cerebral hemisphere can disturb the power of speaking, reading, and writing, without producing any other severe loss of intellectual capacity. Evidently there must exist a group of functions indispensable for language in its widest sense, but not equally essential for all intellectual performance; it is these functions which form the subject of this investigation in as far as they can be affected by destruction of certain parts of the brain.

An organic lesion can only cause a disorder of speech by disturbing underlying physiological processes; the structural changes prevent the orderly performance of certain cortical functions, and speech suffers in consequence of a disturbance of vital processes, which may never directly influence consciousness. These abnormal reactions are apparent to the external world in terms of a disorder of language, just as some defect in the mechanism of a clock is manifested by failure to keep correct time. The physiological activities of certain portions of the cerebral cortex are responsible for the capacity to make use of language in a normal manner; when they are imperfectly carried out, from whatever cause, the acts of speaking, reading, and writing become more or less disordered.

Before we can determine anatomically what parts of the cortex are responsible for these manifestations, it is essential to discover the

nature of the disorder itself. Unfortunately the actual phenomena of disordered speech have been dismissed for the most part in a very summary manner, although an immense amount of time and energy has been expended on the distribution of the structural changes. Clinicians assumed that the defects in the use of language corresponded to three more or less independent groups, loss of speaking, reading, and writing; these in turn were attributed to destruction of "motor," "auditory," and "visual" images. On the other hand, to Marie and his followers loss of speech is in reality a high-grade change in the mechanism of articulation, whilst all other "aphasic" manifestations are attributed by them to a lowering of general intellectual capacity.

This chapter is devoted to considering in how far the actual nature of these disorders of language corresponds to the categories generally laid down. I shall attempt to show that they cannot be classed under the headings of speaking, reading or writing; for, whenever the structural changes affect one half of the brain only, disorder of one of these "faculties" is accompanied by some analogous change in the others. Then I shall bring evidence to show that such affections of language are not based on a destruction of "images." They obey the law laid down by Jackson fifty years ago, that the negative manifestations of a lesion are expressed in some disorder of the affected function; in this instance they appear in terms of language in its widest sense, and not as a disturbance of images, "motor," "visual" or "auditory."

Finally, I shall examine the suggestion that the "motor" aspect of aphasia is in reality an "anarthria," and shall bring evidence to show that this is not an adequate explanation. But the full consideration of the second half of Marie's theory must be postponed to a later period, when I consider more fully the effect on general intellectual capacity of the disorders of language which I am about to describe.

§ 1.—*Disorders in the use of Language, due to an Unilateral Lesion of the Brain, cannot be classed under the Categories of Speaking, Reading and Writing.*

Had it not been for the trend of scientific thought at the time when the facts of "aphasia" were discovered, no one would have imagined that a lesion of the brain could affect exclusively such complex acts as speaking, reading and writing. But it was assumed that man had been endowed at his creation with certain "faculties," situated in different

parts of his brain, and these were thought to be disturbed by the cerebral injury.

This theory seemed to receive support from the fragmentary and insufficient examination of the earlier observers. The patient was said to be able to read and write although he could not speak. Sometimes, on the other hand, he could speak but could not write. But as observations accumulated and the first novelty of the discovery wore off, it became obvious that pure instances of "aphasia," "alexia" or "agraphia" must be extremely rare, and most patients were said to show a "mixed affection" of speech.

From 1866 onwards Jackson opposed such conceptions, and laid stress on the importance of recognizing that there was no such thing as a "faculty" of language. In the same way he insisted that there is no "faculty of memory" apart from the things remembered. But although the doctrine of human faculties may have passed out of fashion, the ideas upon which it was based are still enshrined in the terms "aphasia," "alexia" and "agraphia." Many still hold that it is possible to say that a man has been deprived of the power to speak, to read, or to write by a cerebral lesion, without specifying the conditions under which these complex activities are impossible. It is therefore worth while to demonstrate how little such terms correspond to the actual clinical phenomena.

These "speechless" patients are not "wordless," for they can swear or even ejaculate appropriately on occasion; a man usually speechless may at times produce a complete phrase. But the words of a speechless patient are not at his disposal for voluntary use; they exist for comprehension and can be called up under emotional stress. Voluntary power is diminished with retention of the capacity to evoke the same movement in a more automatic manner. A patient who was unable to find the name for "ink" finally said, "That's what I should call a china pot to hold ink." The disturbance of voluntary speech had destroyed the power of finding the more selective answer "ink," but permitted a more descriptive response. In considering the power to use words we cannot say that the patient is totally speechless; we have to determine under what conditions speech is or is not possible.

In the same way a man may be apparently unable to read a single word, but when a series of printed names of colours are placed on the table, he may be able to point to the one which corresponds to a coloured object in front of him.

The majority of those who are said to be suffering from "agraphia"

can write their names and addresses. One patient under my care wrote without difficulty his name, followed by the full address of his home including the county, but he could not do the same for his mother with whom he lived. In this case the words could be written, when they applied to himself and were more nearly automatic, but not in connexion with another person.

We are face to face with exactly the same difficulties when attempting to determine the power of appreciating spoken words. What, for example, is the condition of a man who cannot draw a "square" when asked to do so, but told to draw a "block of wood" at once draws a perfect square?

Obviously, therefore, we have no right to be satisfied with the statement that the patient cannot speak, read or write. It is our business to discover the conditions under which he can or cannot perform any of these acts. As soon as we examine the clinical phenomena from this point of view, we find that no one of these three categories of language is affected alone by any unilateral lesion of the cerebrum. A disturbance of one aspect of speech is associated with some other disorder in the use of verbal symbols.

This is true even of the form usually called "motor aphasia." For although at first sight it may seem as if the difficulty was confined to the act of speaking, closer observation shows that other functions are also disturbed. For example, in No. 17, writing revealed the same kind of defects as speech; even when well on towards a full vocabulary he addressed a letter to his "Bank Man-ger" and, when silently recording the names of geometrical figures, wrote "pymared" and "pymerad," an exact reproduction of his method of pronouncing these words.

Jackson pointed out the close association between speaking and writing, insisting on their coincident variation. He thought of internal speech as words that had not passed over the vocal organs, and he used writing as a means of testing its condition. Now, internal speech in this sense plays an essential part in many other acts besides speaking and writing. When the patient, sitting opposite to me, attempts to imitate movements of my right or left hand brought into contact with one or other eye or ear, internal verbalization occurs as a phase of the normal act. No word may be uttered, but the words "right" and "left," "eye" and "ear" are essential to correct imitation of this kind. Such imitative acts may, therefore, suffer profoundly in cases of so-called "motor aphasia" [*vide* Table VI, p. 104]. For the same reason these

patients may find considerable difficulty in carrying out the hand, eye and ear tests, when the command is pictorial, although they can execute them correctly when asked to do so by word of mouth. Their difficulty is in evoking the words they require, and verbal command supplies the necessary want.

Much has been made in the past of "agraphia" as a nosological entity. But, provided the lesion of the brain is unilateral, inability to write is always associated with some other loss of function. This is particularly evident when the power of naming is defective; the patient not only finds difficulty in writing down the time as part of the clock test, but he also fails to set the hands correctly to verbal or to printed command. Even in those cases where word formation, naming and reading aloud can be carried out perfectly, writing may suffer badly, and the patient may be unable to execute verbal, printed or pictorial commands.

Thus it is impossible to classify the disorders of language produced by a unilateral lesion of the brain as exclusive affections of speech, reading or writing. For this reason, such words as "aphasia," "alexia," and "agraphia" should never be employed to record the actual phenomena in any instance of speech defect.

§ 2.—*These Disorders of Language are not due to Destruction of Images.*

In 1879, at a time when everyone assumed that destruction of auditory and visual images was a sufficient explanation of the phenomena of aphasia, Jackson recognized that images, together with those unconscious processes on which they depend, might remain intact in the speechless patient. He cannot speak, he cannot write, he cannot read, not because he has lost "images" or "memories" of words, but because he has lost the use of words in speech. In cases where images are disordered, there is a peculiar defect which Jackson called "imperception." Speechlessness may exist without imperception, and it is from such instances only that we can obtain a clear analysis of the processes which make up speech.[1]

I could bring forward innumerable examples from my records of the truth of these statements. In fact, it is difficult to understand how any observer who had studied patients with these disorders of

[1] I hope to consider in a further communication the clinical manifestations of "imperception" and "mind-blindness."

speech or had listened to their own account of their disability, could ever have supposed that "images" were destroyed. They repeatedly insist that they can "see" the dog you ask them to draw, or the room they are attempting to describe. Moreover, most of them have little difficulty in drawing from memory an object that has been shown to them and then removed from sight.

This dissociation between visual images and those activities of language which have usually been attributed to them is well shown by asking the patient to draw a rough plan from memory of some room, such as his hospital ward, with which he is familiar. In many cases, he is entirely unable to do so. But if the observer places a mark on a sheet of paper to indicate the bed on which the patient habitually lies he cannot infrequently indicate correctly the site of windows, doors and fireplace, together with the position of the familiar objects by which he is surrounded. Visual images are intact, but they cannot be represented in the conventional manner. In the same way, No. 2 produced to command the most fantastic and absurd representation of an elephant, although his spontaneous drawings of animals were remarkably good. Images were present but they could not be reproduced to order. The same patient could play an excellent game of chess, which in his case seemed to depend on a strong power of visualization; cards, however, puzzled him and he was quite unable to score.

Auditory images were supposed to be responsible for "memories" of words and they were said to be "stored up" in certain areas of the cortex. But this hypothesis is entirely incapable of explaining the phenomena of aphasia. An officer, who in consequence of a bullet wound of the head had lost the power of speaking, reading and writing, kept beside his bed a list of his possible wants. He would ring his bell and, when the nurse arrived, point without fail to the name of the object he required.

The elderly man, whose condition is described on p. 115, could not speak, read or write. If a card bearing the name of a colour was placed in his hand he was unable to read it: the letter conveyed nothing to him. In the language of the older theory he had "lost the memory for words." But if eight cards, each bearing the name of a colour, were placed on the table in front of him, he could pick out the one which corresponded to an actual colour shown to him.

It is useless further to labour this point. For there is not a single manifestation presented by the defects of language, due to a unilateral lesion of the brain, that can be explained by destruction of auditory or

visual images. The theory had its birth in the study and is contrary to the facts of clinical observation.

§ 3.—*The "Motor" Aspect of these Disorders of Language is not a pure "Anarthria."*

In 1906, Pierre Marie broke away from all the orthodox views of aphasia and propounded the theory that the "motor" aspect of disorders of speech was due to "anarthria," a high-grade disturbance of articulation. On the other hand, he held that the "aphasic" aspect was nothing more than a manifestation of diminished general intelligence. "Il n'y a rien d'aphasique," he says, "dans le trouble *moteur* de l'anarthrique. L'anarthrique comprend, lit, écrit. Sa pensée est intacte, et l'expression en est possible par tout autre moyen que la parole, le langage intérieur n'étant pas altéré."

This statement had the advantage of clearing the ground of older preconceptions, but unfortunately substituted a theory which does not agree with the results of more extensive clinical observation. Marie contends that the anarthric patient, in his sense of the word (or the "motor aphasic" as it was the custom to call him), understands, reads, and writes; his thought is intact, he can express himself by every other means except words, and internal speech is unaffected.

Now it is true that such patients may be able to read and write; but the statement of Marie and his followers implies that these acts can be performed perfectly, and that emissive speech is alone disturbed as a consequence of the brain lesion. Closer observation shows that this is not strictly correct. Amongst my military patients were three who would have been spoken of as uncomplicated examples of "motor aphasia"; they could read and write, and showed no defect of general intelligence or memory, and would so far correspond to Marie's definition of anarthria.

Further investigation, however, showed that other functions were affected besides emissive speech, and this was borne out by the statements of the patients themselves. No. 17 talked slowly and was frequently at a loss for a word; he said: "I sometimes — — have to — — alter the whole — — to alter the sentence — — because — — I — — have — — difficulty — — in finding — — the word." Often he substituted some more picturesque expression, such as "dig it up" for "remember." "I want to say a word — — at the back of my mind — — I can't just — — dig it up." He added,

"At first — — I — — don't think — — I'd more — — than a twenty-word — — voc — — abulary." When saying the alphabet aloud he stumbled over "R"; this he explained by saying that, when he had trouble over "R," it was not that he did not know it, but he wanted to say "A" instead of "R," because he was thinking of "are." If he was not certain of a thing he wrote it down; "I may write it wrong — —, but when I see it — — I know it's — — wrong."

He recognized that he frequently found difficulty with adjectives. "I said a person was strong-willed and strong-headed, and I knew it was not what I wanted to say; half a day afterwards I said, 'You wanted to say headstrong.'"

He could understand what he read if he went slowly, and he never misinterpreted the meaning. But he confessed: "I can't read a book to myself because I'm bothered when I say the words. I can get the meaning of a sentence if it's an isolated sentence, but I can't get all the words. I can't get the middle of the paragraph; I have to go back and start from the preceding full-stop again."

When asked to read a few lines from a book to himself and to write shortly what he had remembered, he did so slowly and with great effort. Moreover, his errors were exactly of the same order as those in speaking. He explained that though he could write he did so with difficulty: "I always have to spell out every word, even the little ones. I have to say 'of'; I know it's a preposition, but then I have to think, Is it 'to' or 'from' or 'of'? Prepositions are always a bother to me."

He was shown a set of geometrical figures and named them correctly, although his pronunciation was defective; he was then asked to write down their names without speaking, and although he never failed to indicate their shape, he wrote "pymared" and "pymerad" instead of pyramid. This closely corresponded to the sort of mistakes he made in pronunciation. Another highly intelligent officer described his disability as follows: "I'm confine (confined) to the words I've got back since my speech came back. I try to make a statement—I have to use the words I've got back—when they say, 'What do you mean by so and so?' I haven't got a further example of what I've said, to explain. My vocabulary (pronounced 'vocab-lery') is still small. When I try to explain I haven't got the words I want, and when they say over a set of words, I then say 'Yes, that's the one.' I have to go over the roots; I trace the root back to the Latin or that, and get it eventually (pronounced e-venchaly)."

Even stronger evidence, that this condition is something more than

an uncomplicated disturbance of emissive speech, is given by the hand, eye and ear tests. The patient sits opposite to the observer and imitates his movements exactly; in this class of case, the task is carried out slowly, and with a varying proportion of mistakes. But as soon as the observer stands behind the patient, and the movements are reflected in a mirror, they are executed perfectly and with great rapidity. An exactly similar phenomenon occurs when the order is given by means of a series of pictures; the patient has much difficulty in executing the movements when he holds the card in his hand, but none when it is reflected in the glass. No. 17 explained his difficulty as follows: "I've always said it is like translating a foreign language, which I knew but not very well. It's like translating from French into English." This patient entirely recovered his speech except for a certain hesitation, and three years later carried out this test in all its forms, without the slightest difficulty.

All these facts and many more, which will be brought forward as examples of the nature of the disturbance produced by these unilateral lesions of the brain, show how little the "motor" disorder of speech can be explained as an uncomplicated "anarthria."

CHAPTER III.—THE NATURE OF THE FUNCTIONS DISTURBED IN APHASIA AND KINDRED DISORDERS OF SPEECH.

No conception that we have so far considered is in harmony with the clinical manifestations of these disorders of language: for any satisfactory hypothesis must be capable of explaining why the patient succeeds in reading or writing under certain conditions, although he fails completely if the task is presented to him in a different manner.

Let us then first of all consider, from this point of view, an ordinary case of so-called "aphasia" with "alexia" and "agraphia." On the evening of May 13, 1911, a man of 56 suddenly lost all power of speaking naturally; next day he was drowsy and the disorder of speech increased. On May 17, he came to my out-patient department, and was admitted to the wards, suffering from severe aphasia without hemiplegia. He was discharged a month later, and remained constantly under the care of Dr. Percy Kidd, until his sudden death on January 6, 1920. His condition throughout this period of nearly nine years remained unchanged; but I was able to make an extended series of observations in November, 1919, and it is on these that the following conclusions are based.

He was almost completely speechless, but could reply "Yes" and "No" correctly: asked to say "Yes," he answered "No, I can't" and told to repeat "No" he usually shook his head, but once said "No, I don't know how to do it." He could at times use the expression "Thank you" appropriately, and when puzzled would say "Can't get it; I know what it is, I try." Once when excited by the request to draw an elephant, he said: "Yes, I've been all over; seen lots! I've got some on my what you call it," alluding to figures on his sideboard at home.

He could not name a single object or tell the time set for him on the clock. But when the hands were placed at 1.30, he held up one finger and bisected it with the other hand; shown 1.55, he held up five fingers and then two, to indicate five minutes to two.

He carried out oral commands excessively badly. Even with the coin-bowl test, he failed in twelve out of fifteen attempts to place the penny named in the right bowl; and yet he had no difficulty in pointing to any one of a series of objects, or choosing the correct colour named to him verbally. It was not words that were lacking, but words used in a certain manner.

Asked to imitate my movements, when I touched the right or left eye or ear with my right or left hand, he failed grossly, sitting face to face. But when my actions were reflected in the glass, he never made a mistake and did not even hesitate. An exactly similar result followed on pictorial commands of the same kind; holding the card in his hand, he was wrong every time, but with the picture reflected in the glass, he carried out the movements perfectly and with great rapidity.

The most remarkable incongruity was shown, however, when he attempted to read. Given a printed command, such as to touch eye or ear with one or other hand, he made no effort to carry out even the slightest movement. He shook his head, saying, "I can't, I know what it is, I don't know what it says." In the same way with the coin-bowl test, he did not attempt to carry out a command given either in printed words or numbers.

But in spite of his inability to execute printed commands, there was no doubt that he could read. He was able to point correctly, on seventeen out of eighteen consecutive occasions, to an object named in print. This exactly corresponded to the results obtained when they were named orally. He was able to carry out an even more severe test on similar lines. The six printed cards of the man, cat and dog test were laid on the table in front of him. Then he was shown

pictures of one pair of the series, for instance, the man and the dog. He picked out correctly, ten times in succession, the card which corresponded to the combination he had seen. Once he chose a card with the inscription, "The cat and the man," but rejected it for one bearing "The man and the cat," the order from left to right in which the figures had been presented to him. Later in the same series, he again chose a card with the right names in the reverse order, but immediately corrected his mistake. Thus, he was not only accurate with regard to the verbal significance of the card he selected, but also paid attention to the order of the words in print.

His signature was reduced to one word, "Beaden" (Beaton), and he was totally unable to write the alphabet when asked to do so; but if it was dictated, seventeen of the letters were perfectly formed and many of the remainder were recognizable.

He could not name a single coin, but indicated the relative value of any two of them by holding up the exact number of fingers. Thus, when a penny and a shilling were laid on the table, he raised his hand with all five fingers extended; said "Nover" (another), held up his hand again and then added two more fingers, making twelve in all. He could even give the relation between a penny and a half-crown, raising his hand six times in succession: and yet he could not count above nineteen, and was unable to carry out simple addition or subtraction.

Asked to draw an elephant, he produced an absurd figure bearing no relation to any living creature. He marked in what appeared to be an eye; asked what it was, he said, "Don't know" but pointed to his own eye. Questioned about a peculiar horn-like projection on the front of the drawing, he caught hold of his own ear; and when I asked "What is that?" pointing to the object hanging down in front, he passed his hand over his own face, moving it upwards, saying, "Sometimes up that way." This was evidently intended to represent a trunk.

It is obvious that this patient had not lost the power of reasoning and was in many ways highly intelligent. Although he was almost entirely deprived of speech and could not name anything placed before him, he could indicate the object or colour named to him orally or in print. He imitated movements extremely badly, and if I sat opposite him or if the order was given in the form of a picture; when however such commands were reflected in the glass, they were carried out perfectly. For, in the first case, the words right and left, eye and ear, must be silently interposed between the reception and execution

of the command; but when reflected in the glass the movements are purely imitative and no verbalization is necessary. When shown a common object of daily use or a colour, the patient had no difficulty in pointing to the corresponding one amongst a group on the table before him. Immediate or intuitive recognition caused him no difficulty.

What then is the function which is disturbed by such lesions of the brain? It obviously cannot be comprised under the headings of speaking, reading and writing; for not only may the loss of power to carry out any one of them be partial, but the disturbance extends beyond their limits and affects other mental activities.

All patients suffering from the disorders dealt with in this paper can choose from amongst a set of common shapes and colours the one which corresponds to that which they have been shown. If a simple geometrical figure or one of a series of objects in everyday use be placed in his normal hand out of sight, the patient can indicate without hesitation the similar one from a group in front of him. He is able to set one clock in imitation of another without difficulty. He can, in fact carry out any operation not demanding symbolic formulation. All forms of immediate recognition are possible which do not require the intervention of some symbol; but all acts of symbolic thinking and expression are liable to be affected, more or less, in the cases of disordered language.

Words are the commonest and most obvious symbols used in thinking, and any action is liable to suffer which demands for its perfect execution any form of verbalization. A gesture which can be accurately imitated, when reflected in the glass, cannot be performed with certainty if patient and observer are seated face to face; for this attitude necessitates translation of the movement into words before it can be carried out. If, however, the command is given in spoken or printed words, it may be executed correctly, because the necessary symbols are provided in the order itself. The patient is not compelled to formulate them, as when he attempts to translate into action gestures made in the face to face position or their pictorial representations.

Any act, demanding for its correct execution a formulated proposition, will certainly be affected, whilst the more closely it corresponds to the matching of two patterns the less it will suffer from these disorders.

This conception almost exactly corresponds to the views put forward by Hughlings Jackson from 1868 onwards. He stated that the words removed in consequence of unilateral lesions of the brain were those

employed in the "formation of propositions"; those which remain to the "speechless" patient are the same words used "non-propositionally" or in the lowliest form of "proposition." Less severe destruction of speech disturbs the use of words in such a way, that the higher and more abstract the "proposition" the more likely is the patient to fail, not only in the emission of a correct verbal equivalent, but in the recognition within himself of the full value of the "proposition." As Jackson expounded this theory in paper after paper, it assumed a form which includes the greater number of the facts I have observed.

It is with the greatest reluctance, therefore, that I venture to change his nomenclature; for I believe that under the uncouth word "propositionizing" is included what I understand by "symbolic thinking." This, Jackson contrasted habitually with what he called "lower forms" of speech or thought. But the question as to what constitutes a proposition is so disputable that it is better to avoid a term which is liable to be misunderstood, and to lead to controversy. Moreover, it is doubtful whether the term is strictly accurate even in Jackson's sense; and it certainly does not cover all the abnormalities observed in cases of aphasia and kindred disorders.

I would therefore suggest that the function affected in those pathological conditions might be called "symbolic thinking and expression." The following chapter is devoted to the various forms assumed by the clinical manifestations, and, after a description of these abnormal states, it will be possible to enter more fully into the nature and limits of this disordered function.

Chapter IV.—Dissociated Forms of Symbolic Thinking and Expression.

In the previous chapter I have suggested that the various disorders of language produced by a unilateral lesion of the brain might be grouped together as affections of symbolic thinking. Every manifestation of this aspect of psychical activity is not, however, of necessity disturbed in any one case: but the more acute and severe the lesion, the graver and more extensive will be the disorder it produces. Sometimes all the specific tests yield an abnormal result; in other instances, if the injury is slighter and more strictly local, many of them may remain unaffected. This leads clinically to the appearance of those dissociated manifestations which gave rise to the conception of various forms of aphasia.

But the consequences of this dissociation are expressed in terms of the function itself, and not as a destruction of auditory and visual images or other processes, which do not form an integral part of the act of symbolic thinking. They belong to the same level of psychical activity, and under normal conditions form correlated factors in the production and comprehension of language in its widest sense. Each of them, however, requires the orderly performance of certain physiological processes, which are disturbed by the destructive or paralysing effect of the lesion. These are not all dependent on the functional activity of the same region of the cortex, and consequently may be affected more or less independently. Each aspect of symbolic thinking is associated more closely with the physiological life of certain parts of the brain, and a local lesion can therefore produce a more or less partial disturbance. The nature of these various defects forms the subject of the present chapter.

But before passing on to describe the actual clinical phenomena, I must utter a warning against the erection of new "types" of aphasia. All the disorders described in this paper are affections of symbolic thinking, and it is this psychical function which is disturbed in consequence of the organic lesion. An exactly analogous dissociation may accompany a lesion of the so-called sensory cortex. All the physiological activities associated with the discriminative aspects of sensation are not uniformly distributed within this area, and a local injury, provided it is not severe, can affect some and not others. The sensory loss is partial; certain tests are affected whilst others give normal results. Such dissociation does not express the elements out of which sensation is primarily composed, but reveals the various forms into which this complex act can be broken up in man. It shows that, with the gradual development of the highest strata of the nervous system, certain parts became more peculiarly associated with the physiological processes underlying particular aspects of sensation. Thus, injury to the sensory area of the cortex may produce complete loss of all sensory discrimination on some portion of the body, or the disturbance of sensibility may be partial, affecting one aspect more than another.

The defects of language which form the subject of this paper follow the same principles. A severe lesion of any part of the brain which gives origin to the physiological processes underlying symbolic thinking and expression may destroy all forms of speech; the patient becomes dumb and cannot read or write. But if the lesion is not associated with widespread disorder of structure or physiological function, some power

will certainly return, and the various tests may not be uniformly affected. Finally, when the period of neural shock has passed away, the abnormal manifestations may be confined, for a time at any rate, to disturbance of some one or more aspects of symbolic thinking. These are not the primary elements out of which speech has been evolved, but the forms assumed when the complete and highly developed function is broken up.

The majority of observers have fallen into the error of supposing that these dissociated manifestations revealed the elementary basis of the acts of speaking, reading and writing. On the contrary, they show the components into which the complete psychical procedure can be split up. At first the whole of symbolic thinking and expression may be rendered impossible, but gradually certain forms return, whilst the recovery of others may be indefinitely retarded. When a man has received a severe injury to his foot, at first he may not be able to walk at all; but after a while he is found to be walking in a peculiar manner according to whether the wound affects his toe or his heel. The gait he assumes is not an elementary component of his normal method of walking; it is due to the fact that he cannot place some one part of his foot on the ground.

Provided we bear these principles strictly in mind, we are justified in recognizing the existence of various forms of aphasia. For the clinical manifestations are so obviously different, according to whether the loss falls mainly on one or other aspect of symbolic thinking, that some differentiation is necessary. But it must be clearly understood that the highest functions are first and most severely affected; tests which are less difficult may be carried out perfectly, although they seem to depend on the same act of speaking, reading, or writing.

§ 1.—*Verbal Defects.*

In severe cases of aphasia the power of using words both in external and internal speech is gravely affected. The patient may be reduced to "yes" and "no," together with a few stereotyped phrases or emotional ejaculations. When due to some lesion originating within the substance of the brain, so profound a disorder of speech is usually accompanied by other defects of language. It is the less severe injuries that reveal a loss of the verbal aspect of symbolic expression in an uncomplicated form.

Amongst my patients with gunshot wounds of the head were three who showed this disability alone. They were unable to produce at will

and without effort the word required, although they could recognize it when it was offered to them either verbally or in print. If they were able to utter the requisite word, they could use it correctly for naming objects, although it might be so badly pronounced as to be scarcely recognizable. They could repeat just as much, or perhaps a little more than they could say voluntarily, and executed without difficulty commands given by word of mouth or in writing. All the remaining forms of symbolic thinking were carried out perfectly and these patients were acutely conscious whether the words they uttered or otherwise used were rightly or wrongly produced.

Let us examine more in detail the manifestations of this purely verbal form of aphasia. External speech is profoundly affected; one patient, who was admitted to the London Hospital five days after he was wounded, could not utter a single word. He rapidly improved and within a fortnight was making sounds which bore a remote relation to the words he was seeking. Both my other patients appear to have passed through this stage, but they came under my care after they had already gained a considerable vocabulary. But it is these slighter disabilities that throw the most light on the true nature of this affection of speech; they not only allow us to study the various forms assumed by defective verbalization, but also permit us to hear some account of their difficulties from the patients themselves. Fortunately No. 17 and No. 4 were highly intelligent officers, who were able to give valuable introspective information.

No. 17 said, "At first I don't think I had more than a twenty-word vocabulary." "I want to say a word, but it is at the back of my mind, I can't dig it up. I sometimes have to alter a whole sentence because I have difficulty in finding the word." He frequently substituted a more metaphorical expression for the usual and more direct word, as for example "dig it up" for "remember." This tendency can be observed in the speech of many normal Englishmen, who are unaccustomed to choose their words carefully.

No. 4, who from the beginning showed a less severe disturbance of speech, said he found difficulty with "tenical terms" (technical terms). "Yesterday I had diff-ulty in remembering what you do with a skull — — Tri — — Tre — — Trephine." "I'm confine (confined) to the words I've got back."

This condition leads to gross faults in pronunciation; No. 6 spoke of "the claration of war by the Ollies" (the declaration of war by the Allies), and all these patients were liable to lapse into similar errors.

These always assumed the character of defective word formation; the sentence might be halting and words were either lacking or mispronounced, but the essential rhythm, both of word and phrase, was intact.

But it must not be supposed that the loss of function in such cases was purely articulatory. Internal speech was profoundly affected; writing showed exactly the same faults as uttered speech. Thus No. 4 said, "At first I used to talk, missing out words; now I don't do that." As a matter of fact this tendency still existed at the time of this confession and was evident when he wrote to dictation. For he complained, "I can't carry many words at a time. If you give me a phrase containing four words I can do three, and then have to get you to dictate further. I find on reading over I leave these small words out, quite often."

When No. 17 was asked to read a short printed passage silently, and to write what he had learnt from it, he wrote slowly, and with great effort. He then said: "I always have to spell out every word, even the little ones. I have to say 'of'; I know it is a preposition, but then I have to think: is it 'to,' 'from,' or 'of'? Prepositions are always a bother to me." "It's not the long words that stop me. It may be words of four or five letters. It's not specially the long words that are the trouble. When I am puzzled over a word like 'help,' I have difficulty in knowing if the l or p come last. My bankers are Holt and Co.; at first I had difficulty in knowing whether the l or the t came last. Now I've written to them so often, I've learnt it." "My spelling of Latin derivatives is better than that of other words."

"I can't spell. I think I can write if I have tried on the blotting paper. I have difficulty in finding the words, because I speak the thing out when I write it. When I write a letter hurriedly, I hurry on and leave out lots of to's and at's and words. The easiest way for me to correct is to get somebody to read aloud, and I say, 'Wait a minute, I want to put in an 'of' or a 'to.'" "I'm teachable. If I spell a word wrong, my wife tells me; I get it right. When I wrote to my bank man-ger (manager) I always spelt 'man' and 'ger.' She told me that; now I get it right." Here his pronunciation of manager was exactly as he had been in the habit of writing the word. "It's no use my trying to spell out the words when I'm blocked with a word. But if I can visualize it, I'm all right; visualize the word as it is written."

The form assumed by written words corresponds in a remarkable way with the errors of external verbalization. In the speechless stage these patients write with extreme difficulty, and may be reduced to

their signature only. As they improve in speech, the power of writing increases, but it tends to show defects analogous to those of pronunciation.

The most striking evidence of the disturbance of internal speech was given by the hand, eye, and ear test (Table VI). These patients found considerable difficulty in imitating my movements correctly when we were face to face. At first sight it might seem as if this had nothing whatever to do with speech. But analysis of our own experience under similar conditions shows that such imitative movements are accompanied by more or less internal verbalization. Although we may not be conscious of saying to ourselves "eye" and "ear," we undoubtedly formulate some such words as "right" and "left"; and this is fully borne out by the patient's own statements.

When, however, he is told to imitate my actions reflected in a mirror, he can carry out the test perfectly without hesitation. For under such conditions the movements can be performed correctly without the use of words. They are direct and intuitive. No verbal symbol intervenes between the reception of command in gesture and its execution.

In order to test the truth of this explanation I asked the patient not to imitate, but to record my movements in writing. This he did badly, sitting face to face as might have been expected from previous experience. But when told to write down my actions, as seen in the mirror, he found exactly the same difficulty. Writing necessitated the interpolation of words into what would have been otherwise an act of non-verbal imitation; this spoilt the response to a test that had otherwise been carried out perfectly. No. 4 explained: "I say to myself, 'left hand'; then I have to say to myself: 'left ear or eye.' When I've said it, when I've decided, I can write it quite easily."

An exactly similar result is obtained in this group of cases, when the order is given pictorially. A series of cards are prepared, each bearing a drawing of the essential factors in one of these movements; these are handed to the patient in order, and he is asked to carry them out without speaking. If he holds the card in his hand, as he would normally look at a picture, the records are bad, just as with face to face imitation. No. 17 said: "It's like translating a foreign language that I know, but not very well. It's like translating from French into English."

But when the figure on the card was reflected in the mirror, his imitative movements were carried out perfectly, and without hesitation.

For no verbal intervention is necessary between the visual reception of the order and its performance.

These patients suffer from a defective power of verbalization; they cannot produce with certainty the word they want either for external or internal speech. They usually appreciate the significance of words presented to them, and can therefore execute both oral and printed commands; but they are liable to become confused in general conversation, or if the order is given too rapidly. No. 4 said: "Some words, when I listen to them, I can't collect the meaning. Nurse said something about your coming this morning; but she said it too quickly, I couldn't collect it; she had to say it again."

An exactly similar difficulty occurs when they attempt to read to themselves. No. 17 said: "I can't read a book to myself because I'm bothered when I say the words. I can get the meaning of a sentence if it is an isolated sentence, but I can't get all the words. I can get the middle of a paragraph. I have to go back and start from the preceding full-stop again."

Reading aloud conveyed the meaning better than when he read to himself; for the sound of the words, though badly pronounced, reinforced his understanding of the significance of the paragraph as a whole.

Writing to dictation is slow, and words may be omitted. No. 4 said: "I can't carry many words at a time. If you give me a phrase containing four words, I can do three, and then have to get you to dictate further—or read it again." On reading what has been written to dictation, such patients notice the omissions, and attempt to correct them. They find no difficulty when the various combinations of the man, cat, and dog test are dictated, and never substitute one word for another, as is so commonly the case in other forms of aphasia.

The verbal aspect of numerals may be affected, but not their significance. Thus, in the early days after the injury the patient may be unable to count, because he has not those verbal symbols which are numbers. With gradual recovery this is replaced by various troubles with compound numbers, and inability to carry a long list of figures in the memory. Thus No. 17 said: "When I first began to look at reference books like the Army List, I couldn't say it was 982 when I looked the page up; but if I didn't try to say it, I could look up the page without difficulty. When I tried to say it I would say different numbers, and then I would muddle myself and have to look it up again. Silent thought was easy, but vocal thought was muddling."

He played an excellent game of bridge, but complained: "When I am scoring in a game, I say 28, when I mean 41. But this makes no difference to my score. I count 41, though I may say 28. I do the sum right, but get the figures I say wrong." Addition and subtraction were correctly carried out, and he kept his own bank-book.

No. 4, however, found some difficulty in carrying over a number when adding or subtracting, although the actual calculations were correctly performed. He had worked in a bank before the War, and was accustomed to add up long columns of figures in his head; this he now found difficult. Such loss of capacity to verbalize figures may lead to actual faults in simple sums if the disturbance is severe and the patient poorly educated [No. 6].

None of these patients failed to name coins correctly if they could find the requisite words; however badly pronounced, the sound bore some relation to the actual name. They could always indicate the relative value of two pieces of money placed in front of them, even if they were obliged to do it in dumb show on their fingers.

Provided they could speak sufficiently, they had no difficulty in naming a series of common objects or even geometrical forms, if sufficiently well educated. They could choose any one of them either to verbal or written command, if the order was given in one word, and not in a complex phrase.

Asked to write the names of such common objects or of colours, without saying them aloud or under the breath, the words produced were recognizable, and corresponded to the object shown; but they displayed the same faults as if they had been pronounced. Thus, pyramid became "pymared," "pymerad"; scissors "sissiors" and ovoid "oboid" throughout one series of written tests.

Drawing is not affected and the outline of a simple model can be reproduced from memory. Told to draw an elephant the result is recognizable and exhibits all the salient parts of the beast. These patients have no difficulty in drawing a plan of some room with which they are familiar; this is a true ground plan and does not show that tendency to lapse into details of elevation so common in other forms of disturbed symbolic thinking. They have no difficulty in finding the way, and can describe a journey from one place to another without difficulty or confusion.

They can look at pictures with pleasure and understanding. They are able to appreciate jokes expressed pictorially or in print, and are capable of playing games at an early stage of recovery. No. 4 found "jig-saw" puzzles very "re — edji — kate — ive" (re-educative).

One of the most striking faculties possessed by this group of patients is the power of recognizing their errors. On one occasion, No. 17 remembered that six months before he had been forced to substitute another word for "economy," which he had read but could not remember. On looking up the actual record it read as follows: "I never found we were much in pocket over her — her — thrift or her — I want another word but I can't get it."

Innumerable examples might be collected of this power of appreciating whether an act of speech is rightly or wrongly performed. It is one of the most characteristic features amongst the manifestations of verbal aphasia.

§ 2.—*Nominal Defects.*

In this disturbance of symbolic thinking and expression, we are not dealing with a difficulty in forming words, but with a disorder of their nominal aspect. A name is a pattern which, if appropriately chosen, fits an external object or state of things around us. When a set of geometrical figures are ranged before me and a cone is placed in my hand out of sight, I know that it resembles one of the objects on the table, and this in spite of a diversity in size, material or colouring. A pattern has been produced as the result of the sensation from my hand, which agrees essentially with what I see. If, however, I give the object a name and call it a cone, I evoke a symbol which indicates that pattern. In the same way, when I give the time as five minutes to eight, I not only utter a series of words, but name a position on the clock; in a few minutes the long hand will reach the hour, and this position is consequently named "five minutes to eight." It differs fundamentally in nominal significance from "five minutes past eight"; the two are as diverse as a cone and a pyramid appreciated by touch.

During the first few weeks, the patient may be almost dumb, with little or no spontaneous speech and unable to write even his signature. But with the passing away of the effects of neural shock he regains his power of speaking. Should he be suffering from a disorder of the nominal aspect of symbolic thought in an uncomplicated form, his enunciation will become almost normal, except where he has difficulty in naming. There is not that profound disturbance of pronunciation which runs through all the utterances of the verbal aphasic.

Thus, exactly two and half months after his wound, No. 7 was unable to name a single one of the common objects presented to him in

order; he moved his lips like a person seeking for a word and said: "I can't say it. I think I know it, but I can't say." In so far as he could find words to express himself they were correctly pronounced. He was unable to repeat these names, although the sound he uttered bore a distinct relation to the word said by me; in some cases it came so close that it might be considered correct. For example, key became "kay," pencil "pintil," matches "mats," and penny was twice repeated accurately. Asked to repeat "scissors" he says "sis—I can't open far enough," pointing to the jaw.

He could not write the names of any of these objects, but he could copy them from a printed card in capitals only; he could not translate the words into cursive handwriting, for this seems to demand a power of consecutive nomination.

When I named one of the objects on the table in front of him, he chose slowly and was evidently puzzled; in three instances he gave up altogether, but in nine out of the twelve observations he made a correct choice. On presenting him with the same words singly in print he failed to select any object on six occasions, but chose correctly, though with hesitation, in the remainder.

When however he was shown one of these common objects or when it was placed in his hand out of sight, he never hesitated for a moment; in every instance his choice was correct and prompt.

This disability does not depend on destruction of images but on a want of names. No. 2, who had reached a further stage of recovery, could choose correctly to verbal command, although his power of naming was very defective. So perfect was his memory of the order in which the objects stood upon the table, that with his eyes closed he could point to each one in turn as I named it; but asked to give their order in words, he was completely unable to do so.

When this patient was told to say the days of the week, the first four were correctly given, but Friday was called "Fysday" and Saturday, "Second—Septen-day—Sutton-day": then after a pause he remarked "Sunday is the only one I can remember after that." Finally, after three attempts he went through the week correctly except that "Second-day" replaced Saturday. He was then given a series of cards, each of which contained the name of one day in print; these he copied perfectly in capitals, letter by letter. His lips did not move and this was obviously pure imitation. When, however, he attempted to translate these printed words into cursive handwriting, he muttered each word under his breath and wrote, "Mondey, Tuersty, Wednersty, Thersteay,

Freray, Satereay, SunDay." Asked to read the printed words aloud he did so with remarkable correctness, until he came to Friday, which was first called "Fys-day" and then "Friday"; he said "Secon-day" but changed it to "Saturday" and finished with "Sunday" perfectly pronounced. When he attempted to write the names of the days of the week to verbal dictation, he failed badly and most of them were left unfinished; Friday, however, he wrote correctly. Told to write them spontaneously, he was equally unsuccessful, although each word began with the appropriate letter. He was then told to repeat the days of the week after me, and to write each one as he said it. His enunciation was perfect, and although he continued to say the word correctly all the time he was attempting to write, the results were little if at all better than when he wrote spontaneously.

Here we see that correct verbal imitation was possible either when repeating the sound of the words or when copying print: but any act which required the intervention of the nominal aspect of symbolic thinking or expression was badly executed. Worst of all was writing to command; for this demanded not only the power of naming, but also the translation of the name into written symbols.

Except in the earlier stages, these patients can read to themselves, but with great slowness and uncertainty. They are particularly liable to grave misunderstandings due to defective appreciation of the nominal value of the words they are reading.

If what they are given to read silently is in the form of a command, this want of appreciation becomes still more apparent; for not only must the patient understand the printed matter, but he is compelled to formulate the meaning to himself before he can carry out the order that lies behind it.

No. 7 was given a series of cards, one by one, each of which contained a simple order such as "Shut your eyes," "Touch your nose," &c. He was told to read it to himself and to "do what it says." This presented great difficulties; sometimes when told to shut his eyes, he brought his hand to his eye, or asked to give his hand, he touched the left with the right. Once only in eight attempts did he carry out such orders correctly. Reading them aloud seemed if anything to cause greater difficulty, and copying the legend on the card did not materially improve his power of carrying on the action demanded. But, as soon as the commands were given him verbally, he made one partial mistake only, touching his eyes with his hand, instead of closing them.

Reading aloud seems to add to the difficulty and is no aid to under-

standing, as with some other forms of aphasia. The patient tries to spell out the words and becomes confused because he is uncertain of the nominal value of the different letters. At a time when No. 2 could both say the alphabet correctly and made no mistakes in writing or reading it, he was unable to read aloud the man, cat and dog series. Given "The man and the dog" in print on a card, he said, "T-H-E.: the next one is M-A day, no D. This is A. This is N. This is — — D T and H and E. This is D. That is O and T. I'm not sure about that one." Asked "What does the whole say?" he answered "The only thing I can tell you is it was a dog." This is a fair sample of a series of observations on his power of reading aloud a combination of simple words not containing a command.

But when pictures of the man, the cat and the dog were shown to him in series two at a time, he never hesitated for a moment but named them accurately. Evidently he still possessed the power of evoking these simple words as symbolic representations of pictures. But when offered the names in print he was confused by want of certain appreciation of the value of each letter. He had no difficulty with the alphabet as a sequence: it was the nominal value of any one of its constituents which was uncertain.

In these observations all the words were monosyllabic and recalled a child's first reading book; much of the difficulty arose from defective comprehension of single letters. The same objects represented pictorially could be translated with ease into their nominal equivalents. But as soon as pictures were made to carry a command, as in the hand, eye and ear test, the action demanded is as badly carried out as if the order had been given in print. The necessity for formulating the significance of the picture gave it a high nominal value.

The nature of this disturbance of symbolic thinking comes out particularly clearly with the clock-tests. There was not the slightest difficulty in setting one clock in direct imitation of the other; even the short hand was moved into a proportionate position between the two numbers of the hours. But none of these patients could tell the time or write it correctly. They set the clock badly to oral command or when the order was given in printed words; but when it took the form of figures, so called "railway time," it was perfectly carried out. For to set a clock when given such a number as 4.45, the patient first places the hour hand at 4, and then sweeps round the other one to the position he associates with 45 minutes; but if he is asked to set "a quarter to five," the temptation is to set the one hand at 5, whilst the

other hovers uncertainly between "a quarter past," and "a quarter to." This difference does not, however, apply when the patient attempts to tell the time; it is equally difficult for him to evoke the name "4.45" as "a quarter to five."

Throughout these tests there is a profound tendency to muddle up "to" and "past." The number of the hour has an overwhelming attraction; for example, when setting twenty minutes to six the long hand may be rightly placed, but the hour hand points to six. Not infrequently the nominal value of the two hands was mistaken and the long one set to the hour.

All this does not signify that the patient does not know the time. No. 2 said, "That is when you eat," or "when we were there," of some particular event, and is always right, although he cannot reproduce the time in either case. Moreover, when shown the test-clock set at some definite hour, he could pick out a card on which the corresponding time was printed in figures, ten times in succession without a moment's hesitation. It is not knowledge of the time that is lacking, but the symbolic means of expressing even to themselves what they know; at the same time they have difficulty in understanding the nominal aspect of words, spoken or printed, in which they are told how to set the clock.

Although it is impossible to name an object with any certainty, the patient may be able to do so by using some metaphorical expression instead of a single word. For example, during the tests with colours, No. 2 was wrong in eleven out of sixteen attempts to name one of them set before him (Table II, p. 97). So bad were his mistakes that he might have been thought to be colour blind; for white was called "green," black "red," and green "blue." Exactly the same kind of error occurred when he chose a colour from its printed name on a card; in this case he even chose white for black, and black for green. When asked to read them aloud eleven out of sixteen were wrongly named.

From the observations we might be tempted to think that he was devoid of all knowledge of the nature of colour, in fact that he was "mind-blind." But in his attempts to explain to me his difficulty in reading the printed cards he began to point to my white coat, his khaki tie, the blue band on his arm, which he wore as a wounded officer, and the green of the trees just outside his window. Instead of the names of the colours, he was encouraged to use a series of similitudes; black was "what you do for the dead," red "what the staff wear," or, pointing to the lapel of his tunic, "where the staff have it"; blue was

"my arm," green "what is out there" (pointing to the trees), white is "what you wear" (a white coat), yellow "this one" (holding his tie). Twelve times he was shown cards bearing the name of one of these colours, and in every case he gave the right answer.

I then reversed the procedure, saying to him "the dead" and he chose a black piece of silk from a set of colours on the table in front of him; for "what is on your arm" he selected blue, "what the staff wear" red, "what is out there" green, "like your tie," which he was not allowed to look at, led to his choosing yellow. For violet, we had agreed on a lamp-shade that stood by his bed; this had been removed from the room before these observations began, but to the words, "like a lamp shade," he chose violet. Throughout he made no mistakes in fourteen consecutive tests. This shows that he had an acute sense of colour, but that names were wanting for expression, and were badly understood when presented, both verbally and in print.

The power of counting or uttering numbers in sequence usually returned step by step with that of saying the alphabet. No. 2 had no difficulty with either when first he came under my care; but this did not mean that he could employ any particular numeral directly and with certainty in arithmetical operations. He could pick up five things when told to do so, but asked to add five to any number, he would count on his fingers, or would place five dots on the paper; the figures 2, 3, and 5 rapidly became associated with two, three and five dots, which he visualized around them; but 4, 6, 7 and 8 were counted on his fingers or dotted on the paper.

He confessed that numbers puzzled him at first; if "eighteen" was said, the meaning did not come until he had counted it up. In the same way, he could say the alphabet perfectly, but the nominal value of any single letter was frequently defective. This difference in the facility with which a name is appreciated in series and as an isolated word is familiar to all of us in a foreign language; many who can rattle off French numerals without difficulty, experience a moment's hesitation when told that the price is "quatre vingt douze francs."

No. 2 experienced the same sort of difficulty when he was informed that some engagement was fixed for "Tuesday the twentieth of March." Telling him the day of the week or the month did not convey to him the part of the week or of the year in which it fell.

All three patients failed to carry out even simple arithmetical exercises. They could add 356 to 231 on paper, but all of them failed over such a sum as adding 275 to 856, which required the carrying over of a

number from one column to the next. Captain C. (No. 2), who was by far the most highly educated, counted up to five, and then on for another six, giving the answer correctly as eleven ; he then counted seven, and one more, making eight ; from this he went on for five more, saying "thirteen." He wrote "3" but was wrong with the last column of figures because he forgot to add the 1. Both the other patients wrote down the sum of each pair of figures separately ; thus No. 7 wrote :—

```
  2 2 8            3 4 5
  7 3 4    and     8 6 8
 ------         --------
  9. 5.12       11.10.12
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All of them broke down completely when asked to do simple subtraction, and No. 7 took the lowest number from the highest, whether it was in the upper or lower line of the sum ; No. 18 always worked from left to right throughout.

As might have been expected, there was considerable difficulty in finding the usual names for coins, although it was obvious that they were still recognized correctly. Thus, a sixpence was called "half a shilling," two and six was "two and a half," a ten shilling note was "half a pound" ; and yet, in a series of twenty-four observations, many coins were given their usual names, and there was no evidence that any one of them was mistaken for another of a different value.

But when the attempt was made to give the relative value of two coins, such as a sixpence and a shilling, laid in front of him, the patient became confused ; he tended to add them, saying "one and a half." With 1d. and 6d. Captain C. said "seven." When, however, the question was put in the form, "How many of that [6d.] would you have to give me for that [1s.] ?" he answered "Two : one more of that [pointing to the 6d.] to get one of that [the 1s.]." Then he suddenly burst out, "One more sixpence to get a shilling." Throughout this series of tests he could never name the relative value directly of any two pieces of money, and yet by these indirect means it was possible to show that he understood perfectly. No. 7 showed exactly the same difficulty in formulating the relationship, but was able to pile up a heap of coins on the one side which exactly equalled the larger one in value. Given a shilling, he collected together a sixpence, a threepenny bit, and three coppers ; a two-shilling piece was placed before him, and he chose from the money on the table a sixpence, a threepenny bit, a shilling, and quickly added three coppers. Yet when asked, "How many of this [6d.] go into that [2s.] ?" he said, "Two — two — two shillun —," and with 1s. and 2s. replied, "Three shillun." But behind

this difficulty in expression lay a correct and intuitive comprehension of the relative value of the coins to one another.

Nearly two years after the injury Captain C. (No. 2) was able to fill up a cheque spontaneously; but he could not be certain that the written words and the figures corresponded with one another. On the occasion when he drew a cheque in my presence for eighty-five pounds, ten shillings and sixpence, he filled in the figures as £80 10s. 6d. He noticed this discrepancy and succeeded finally in making the correction; but the cheque would not have passed the bank.

All these patients could draw from a model, and, after its removal, reproduced the drawing from memory. But when No. 2 and No. 7 were asked to draw an elephant, the result bore no relation to this animal; it was without trunk, tusks or ears. This was the more remarkable as Captain C. (No. 2) used to amuse himself by drawing pictures of the animals he had shot in Cashmir; on one occasion, when talking about transport in the East, he drew spontaneously a spirited rough picture of a camel. Visual images are not destroyed, but they cannot be evoked with certainty at will or to command.

This is particularly evident when the patient is asked to draw a rough plan of some room he knows well. No. 7 was asked to put down on paper the relative position of the objects in the ward he could see from his bed; he failed completely. But when I drew an oblong on the paper, saying, "That is your bed," and asked him to point to the situation of the various things he could remember, he was astonishingly accurate. For instance, he pointed to the left, saying, "There's only my bed," and to the right, "There's a bed; no one there now"; he then indicated the head of the bed, and touched the wall of the examining room in which he was at the time, to signify that here was a wall. All these details and many others were correct, but he could not put them down himself on paper. Captain C. (No. 2) started to draw a plan, but filled it in with details in elevation. Images were certainly intact, but they could not be reduced to a symbolic formula.

Orientation is unaffected and such patients have no difficulty in finding their way, provided they are not asked how they intend to go from one place to another. No. 2 succeeded in explaining this to me as follows: "I have no streets in London; no names at all. Suppose I was going from the Army and Navy, that's what it's called, just round here (the Army and Navy Stores), I should then say some place to a hospital about a quarter of a mile away. I remember the hospital is

on the left on the way to the War Office, about half-way. I believe it's the Abbey. No, it's near the Abbey on the way to the War Office." Later he added, "I saw the Hospital." I put as a question, "The Abbey?" and he replied as follows: "It's here, but then it's gone again and I have to feel for it again. The only thing I can remember of it is the opening, the big opening, where everybody goes in. I can get that" (moving his hand in the form of a large arch). Since it was obvious that he had described Westminster Hospital and Westminster Abbey, I asked, "Have the Hospital and Abbey anything to do with one another?" He answered, "Nothing except my focus, the place of them; the distance, that is all. I should say how far the Hospital is to the Abbey in re- —, in re-, — that's where I go wrong. I want to say in re- something [relation]. There are just little bits in expressing what I want to say; little bits in which I have to turn my brain another way to get what I want to say, whereas a year or two ago I should have said it without thinking. You see, it's like this: with me it's all in bits. I have to jump like this," marking a thick line between two points with his pencil. "Like a man who jumps from one thing to the next. I can see them but I can't express. Really it is that I haven't enough names. I've got practically no names. The easiest thing is what I do now. I say what I can, it's all wrong; but they get an idea of what I want to say."

It is evident from this conversation that he was able to recall visual images of objects on his way from the Empire Hospital in Westminster to the War Office in Whitehall. First of all, he saw the Army and Navy Stores and then Westminster Hospital on his left, with the great door of Westminster Abbey on the right; but want of names prevented him from connecting the two, except in position, and he was forced to jump from one image to another without the cohesive links of verbal formulation.

Pictures apparently conveyed their full meaning so long as they did not imply a command to carry out some action. No. 18, when shown a picture of an aeroplane looping the loop, explained the manœuvre by pointing and gestures, helped out by words. No. 2 saw all the points in a Bairnsfather picture of "Old Bill" boxing with Hindenburg. But jokes frequently present considerable difficulty if they demand nominal formulæ for their comprehension.

These patients enjoyed games and could play dominoes and draughts. Captain C. (No. 2) was above the normal average at chess, but could

no longer play bridge. "The names of the cards bother me," he said. "It's just names; I used to play a good game of bridge."

§ 3.—*Syntactical Defects.*

This form of disturbance of language can be distinguished by the fact that the patient talks jargon. In either verbal or nominal aphasia he cannot easily evoke the words and names he desires to use, and consequently speaks slowly and with difficulty. In this group, on the other hand, he talks with great rapidity when once started; the words may be recognizable but badly put together, or, if the loss is more severe, they may be pure jargon. The rhythmic movements of the phrase are affected; they are hurried and slurred, and the patient cannot "touch off" the words so as to produce a correctly coherent sentence. In the lighter forms of this defect there is no difficulty in finding words or names, which may be intelligible, especially if the subject of the conversation is known. Thus, when No. 15 was given quinine as one of the tests for the sense of taste, he said, "Rotten to drink it. Something medicine or that. Make you drop of water after it, so to take out of your mouth." Sometimes speech closely resembles baby language; for example, asked what his right hand felt like, No. 13 said, "Tiff-rent from uffer 'n — ka tell ooh, know zis un seems strong."

But at times even this patient became unintelligible; describing to me a picture of a tramway car with a woman at the wheel and a man conductor he said: "Here's lay, here handle, the man conduker, on the nines, shot steats on it, zee passengers, two man, lady" (Here is the lady; she's at the handle; the man conductor. On the lines; it's got seats on it. Three passengers, two men and a lady.) He then read the the legend at the top of this picture as "Mins bixet o-er men" (Woman's victory over man).

The power of naming an object in sounds that are comprehensible varies with the severity of the affection. Thus, in No. 13, single words could be produced perfectly as names so long as they were applied to familiar objects; but when attempting to name colours he "jargoned" orange, green and violet; in every instance, however, the sound was recognizable as a name. No. 15 was extremely unstable in his nomenclature, using "blacking," "pencil" and "black-lead" indiscriminately for pencil. In naming colours he also found difficulty with orange, violet ("volley") and to a slighter extent with green; but all the truly monosyllabic colours he named correctly.

But in No. 14 the sounds, though emitted volubly, seemed to bear no

relation to the name of the object ; thus, matches became "Stes-miss-ness." He then seized a pencil and wrote "match," showing that he knew the name. With a penny he shook his head, said "No" but wrote the name correctly ; in some instances, however, he could neither say it intelligibly nor write it at all. Here the jargoning had become so severe as to destroy all power of producing some names at will.

He could say "yes" and "no" correctly, and answered "Come in" to a knock at the door. He was extremely intelligent in the manner in which he employed his jargon, and was particularly fond of using "there" and then pointing to the map, the book or the picture to which he wished to refer. He used words or short phrases, but could not amplify or repeat them at will. On September 15, 1915, he was reading a French account of Napoleon's Russian campaign, and wanted to tell me how closely the dates coincided with the Russian retreat which was then taking place. He took the book in his hand, pointed to the date "15 Septembre," saying, "Now — just the same — there — yes — over there."

One day during a set of observations he said, "Funny thing — this worse — that sort of thing." Then he seized his note-book and wrote, "as, at" ; I asked "You mean conjunctions and prepositions?" and he replied, "Yes — that sort of thing."

The grammatical structure of the phrase is liable to be badly affected. The patient talks fluently in short jerky sentences, slurring or omitting many of the junction words. Even when they are present, it is difficult to hear the articles, prepositions and other components of syntax. Asked what he had done since his admission into the London Hospital, No. 15 said, "To here—only washing—cups and plates. That's about all you've got to do here." "Have you played no games?" "Played games—yes—played one, daytime, garden."

The power of writing suffers on the whole less than external speech, because the defect is essentially one of balance or rhythmic utterance. Thus No. 14 made unintelligible sounds when asked to name a set of common objects lying on the table before him ; but in thirteen out of eighteen serial attempts he succeeded in writing names for them, which were recognizably correct. Single words used as names were so much more easily written than spoken, that this patient always carried about paper and a pencil to help him in his conversational difficulties.

After the acute phase of shock has passed away, it is always possible to obtain some recognizable signature, and usually the address can be correctly written. But No. 15 who wrote his name and address

perfectly could not write that of his mother, in whose house he lived; the unfamiliar beginning inhibited the almost automatic remainder of his action.

Patients with a severe degree of this disorder cannot write a letter, but in the milder cases such spontaneous writing is carried out far better than might have been expected from the character of external speech. This is evident when the patient is asked to read what he has written; No. 13 wrote a perfectly coherent and well-spelt letter, but jargoned badly on attempting to read it aloud.

The power to write from dictation shows all grades of disturbance from almost complete loss in No 14 to a difficulty with consecutive phrases, but not with isolated words or sentences. Thus No. 13 succeeded perfectly in writing the man, cat and dog series to dictation, but failed badly with a passage chosen by himself from the newspaper.

All these patients could copy correctly and transcribed print with cursive handwriting without hesitation.

Reading to themselves was their favourite recreation and, although they showed evidence of remarkable powers of comprehension, they were hampered by the disorderly structure of the phrase. No. 15 confessed, "Don't always know what they mean going to talk to." No. 14 followed all the movements at the Front on large scale military maps, and enjoyed demonstrating to me the changes reported day by day in the newspapers. But any attempt to impart this information verbally revealed his disabilities in an acute form. Moreover, he was quite unable to follow up a conversation suggested by what he had read to himself and understood. After he had pointed to "15 Septembre" in his French book on Napoleon's Russian campaign, I answered, "Yes, that was also a beautiful summer and the snow came early." He replied, "Yes—oh! did it?—oh! yes." He showed so much interest that I asked him when Napoleon first had difficulty with the snow; but he turned the pages aimlessly. I then questioned him as to the date on which Napoleon reached Moscow (September 14, 1812), the starting point of our conversation, but he shook his head and could not answer. Spontaneous thought was rapid and correct, but the power of reproduction was evidently defective.

The following incident in his life whilst under our care shows how accurate may be the processes of intuitive comprehension even with a severe syntactical disturbance. He was walking in Kew Gardens with the medical officer in charge of the Hospital, when he pointed out

a new variety of heath. The doctor said, "Scotch," to which the patient answered, "No, no, you and me." It was an Irish heath and both the patient and his companion were Irishmen.

The nature of this disturbance of symbolic thinking was clearly demonstrated by a series of observations carried out with No. 13. He chose a picture from the daily paper showing a box for the receipt of flowers for wounded soldiers, placed on the platform of Snaresbrook Station. Underneath was the legend, "This box has been placed on the platform at Snaresbrook Station as a receptacle for flowers for the wounded. Many of the passengers contribute nosegays daily from their gardens, and these are forwarded without delay to the Bethnal Green Infirmary." As a Londoner in an East End hospital this interested him greatly.

Asked to write down what he had gathered from his reading and from the picture, he wrote, "At Sanbrook station they have large box which are collicting flowers for the wounded soldiers, and they are sending to the Velnah Green Hospital." I dictated the printed description with the following result: "This box has been placed on the platform a smatbrook station as sesful for the wounded many of the passengers contic nonsgay from there garndens and these without delay to the Belnelth Green imfirmary"; but he was able to copy the printed sentences in perfect handwriting without a fault or omission. Given the paper and asked to read it aloud, he produced the following jargon: "Zis box had been place on the plakform at Senbrook Station as a . . . for flowers for the wounded . . . Many ob le pasn-gers contibute nosezays from their gardens and these are for-boarded without delay to Besnal Green Internary. . ." Shown the picture, he pointed out all the various significant objects without hesitation, giving them recognizable names. "Box, Flowers for wounded shoulders. On the back, plakform, Senfbrook. Ladies see, putting flowers into box."

He evidently understood the significance both of the printed legend and of the picture, but he could not reproduce his conception in coherent words or in writing, although he could copy correctly.

In the more extreme cases, disorders of expression make it impossible to test the power of reading or writing from pictures. With a less severe disturbance, however, a simple test, such as the man, cat and dog series, can be carried out, when two of the three pictures are presented to the patient together. But the response, both oral and in writing, tends to take the form of the correct names with no junction words. Thus, No. 13 had been through the variants of this test in

which he had read aloud and had written to dictation a series of combinations of the man, the dog and the cat. But when shown simultaneously any two pictures, for example, the man and the cat, he replied systematically, "Man, Cat," &c. After this series, expressed in spoken words, he was again shown the pictures and asked to write silently what he had seen; he wrote "Man, Cat," without articles or conjunctions.

Another peculiarity of this set of answers was as follows: The normal man when shown two such pictures, that of the man to the left and the cat to the right, frames his answer as if he was reading, and says, "The man and the cat." But No. 13 showed a remarkable inconstancy, both when giving his reply in words or writing. Sometimes he placed the left hand figure first, sometimes that to the right. This inconstance of direction appears again in some of his answers to the clock test given in detail later.

These patients can count and carry out simple addition and subtraction perfectly. No. 14 could make out a cheque for the correct amount spontaneously; but when told to fill it in for a certain sum he inserted figures in the place of the words and wrote, "Five pound 10/6" where he should have written numbers only.

All these patients could recognize the relation between two coins placed on the table in front of them, and none of them experienced any difficulty with change.

The clock tests proved unusually interesting, because, according to their results, the three patients could be arranged in conformity with the known severity of the disturbance. In every instance the clock was set correctly both in imitation and to printed command; to oral command No. 14 made gross errors, No. 15 was slow, but No. 13 made no mistakes. On attempting to tell the time No. 14 failed in every attempt; No. 15 made two errors in substance, but adopted an extraordinary nomenclature; No. 13 was slow but substantially correct. Telling them to write down the time set on the test-clock led to identical results, and No. 15 showed the same extraordinary method of recording the time as when telling it aloud. At one time he wrote 45 to 9, at another 20 past 11; he called 8.10 "2 past 8," because the long hand, pointing to ten minutes past the hour, stood over the figure 2. A similar instability of nomenclature was shown throughout this case, whether he was giving names to common objects and colours or telling the time in speech or writing.

Drawing from a model, or from memory of the same object shortly

after it had been removed from sight, was not affected; in fact No. 15 produced a remarkably successful picture of a spirit lamp and said, "I was a good drawder — drawer — at school." But when he was asked to draw an elephant, which he had seen during his service in India, the outline of the body somewhat resembled this animal, but was without trunk or tusks. He proceeded to fill in the mouth, saying the word aloud; then he said "Highons," "Irons" (horns) and immediately added horns to the drawing. After he had finished I asked, "What has an elephant got in front?" He answered, "They carry big trees — — tied round a bit of an iron thing." I then said, "Behind you have given him a tail, what has he in front?" He replied, "He has a big one, quite straight, about a yard long." "What is it called?" "Same what you drink water with." "Has your elephant got a trunk?" "He's lost it," and at once he wanted to add the trunk.

I then pointed to the horns he had drawn on the head of his figure, asking, "Has an elephant got horns?" He replied, "Yes, silver, what you stick out" (pointing to the corner of his mouth and placing his pencil into the position of a tusk). "What are they made of?" "Kind a white bone one, what grows in the mouth — — on the roof — — on the edge of the mouth." Evidently he was confused by saying the jargon words representing horns, and impulsively added them to the figure; but he was finally able to explain his error by gestures giving a correct interpretation of his intention.

No. 13 was more successful, but added the trunk afterwards, saying "Tump."

None of these patients had any difficulty in finding their way, or in pointing out from memory the position of the principal objects in the ward.

They enjoyed games, and both the privates played dominoes well.

They seemed to recognize that the words they uttered were inadequate to express their meaning. Thus, when No. 15 talked jargon so badly that I could not understand him, he drooped his head, blushed and laughing sheepishly did not care to try again to express that particular thought. He was intelligent and useful in the ward, waiting on the other patients, washing up and laying the table correctly for meals; but as soon as he attempted to formulate and express some want he became confused and could not make himself understood. No 14 helped himself out of his conversational difficulties by writing single words in a notebook which he carried about with him. Any gross failure to make himself under-

stood produced an outburst of intense irritability; this was calmed immediately by giving him some test he could perform with ease.

This patient was musical and played me Chopin No. 20 Largo very slowly, reading the notes and giving the change of key correctly. The right hand was clumsy owing to the cerebral injury; but he succeeded in bringing his fingers on to the right notes of the chord, and if he was wrong immediately corrected his error. Keeping his eyes on the music, he recognized by ear when he had struck a false note and that it did not correspond with the text of the music. He played to me other pieces of Chopin, correcting the faults due to the clumsiness of his right hand; but the slow pace of the Largo was in his favour, whilst the complexities of the change in key showed how clearly musical notation conveyed to him the notes intended.

§ 4.—*Semantic*[1] *Defects.*

I have described the various changes in speaking, reading, and writing, produced by affections of the verbal, nominal, and syntactical aspects of symbolic thinking and expression. One other form of disorder emerges from analysis of the clinical phenomena due to unilateral lesions of the brain. This may be called "semantic," because it is comprehension of the significance of words and phrases, as a whole, which is primarily affected.

This has usually been spoken of as an "amnesia," and the patient is said to have lost "the memory of words." But there is no such thing as a "faculty of memory" apart from things remembered; a man who has lost his "memory for words" from a local and unilateral lesion of the brain is suffering from an affection of speech. This may be of the same order as the disturbances of word formation we have considered under the terms verbal, nominal, and syntactical aphasia. The difference lies in the fact that in this case the defect is manifested by an inability to appreciate and retain the full significance of words and phrases.

These patients suffer from no difficulty in pronunciation; intonation and syntax are perfect. They can name common objects correctly, and indicate the one that has been mentioned orally or in print. But

[1] From σημαίνειν, to signify. This term was used by Bréal in his well-known "Essai de Sémantique," a study in the science of the ultimate meaning of words. Since I am dealing here mainly with a loss of power to comprehend the full significance of words and phrases together with a want of capacity to use such modes of expression, I have not hesitated to adopt this term, which has already become part of the English language.

when similar experiments were carried out with colours, the answers were less certain, and the patient showed some hesitation and confusion. Oral commands might be badly executed, although the choice made in answer to the printed name was correct. Moreover, he did not seem to remember the relative position of the coloured materials on the table before him; when shown a colour on a printed card bearing the name, he passed his hand backwards and forwards over the row of specimen colours before him until he found the one he recognized. Normal persons rapidly learn the order in which the eight colours lie on the table, and, after the first few experiments, go straight to the one which matches the pattern without groping. This also occurs both with verbal and nominal aphasia; in the latter condition I was able to show that the patient retained a perfect memory of the order in which the colours lay, although he could not name any of them with certainty.

The nature of this disorder of symbolic thinking is clearly revealed by the clock-tests. In none of the other forms was there the least difficulty in setting the hands of one clock in strict conformity with those of another; but all the patients of this group carried out this manœuvre slowly, with hesitation, and one of them showed definite evidence of confusion. They could tell the time correctly, but when asked to set the clock in response to either oral or printed command, the errors were extremely gross. The long hand was set as if it was the short one; "to" and "past" were mistaken for one another, and even the hour was wrongly indicated in some instances.

Since the patient could tell the time correctly, his inability to set the clock must have been due to some want of apprehension of the significance of a spoken or written order. No. 10 confessed: "If I can't get the hands exactly where I want, I lose grasp; I get thinking"; and No. 8 said: "I can't make out the difference between *past* and *to* six. I don't know from which side to approach it."

Here we find expressed that inability to recognize significance or to appreciate intention, which is at the root of these semantic disorders. It is not surprising, therefore, that the hand, eye, and ear test was extremely badly executed, necessitating as it does accurate choice between three pairs of possible actions. Moreover, most patients with other disorders of symbolic thinking recognized, when the observer touched his left ear with his right forefinger, that such a movement implied crossing of the hand to the opposite side of the face. One of the most characteristic errors of the semantic group was due to want

of appreciation that the action was crossed. Eye and ear might be mistaken, and the patient was particularly liable to give up before completing the action. No. 10 said: "My initial difficulty seems to be right and left; it confuses me; I forget the rest of what I've got to do." Even in the selection of the right or left hand he was obliged to employ a "memoria technica"; "I clench my fist, and then I think of boxing, delivering a blow with the right or left hand; but then, in touching the eye, that feeling is gone."

In all normal persons and with other disorders of symbolic thinking, movements reflected in a mirror can be imitated with much greater ease than when the patient and observer sit opposite to one another. This also applies to the same actions, when given in the form of a pictorial command. But in the semantic group, though the number of errors may be less when the required movement is reflected, there is not the conviction, so characteristic of most of the other disorders of symbolic thinking, that the action is extremely easy. No. 8 said, "When I imitated you I had to think: is that his right hand or his left? When I'm looking in the glass it is easier because the sides are the same; but then I begin to think it out and get puzzled." After he had made an extremely bad record to pictorial commands reflected in the glass, he explained, "Somehow or another I didn't seem able to get the right part of the picture; sometimes I seem to look at one, sometimes at the other. I have to reason out the meaning of the whole picture."

Everything tends to be appreciated in detail, but the general significance is lacking. This is evident when the patient is given a picture, and told to say what he sees in it. He looks at it like a child, pointing out one thing after another, and not uncommonly misses some important feature; asked what it means, he may be entirely at a loss and may then invent some preposterous explanation. No. 5, who was a gardener from the country, chose from the paper the picture of a man riding a cow, over which stood the legend, "Mayor's Curious Steed." He said, "That's a man riding on a colt. No, sir, it isn't, sir, it looks more like a cow — — or a young cow — — no, it isn't — — heifer, sir." After he had read the legend, I asked, "Who is the man?" and he replied, "A farmer, sir. No, Major — — No, the Mayor curse sted — — the Mayor curious stid. It's something you don't see every day, that stid; something very uncommon that animal. It's in a horse's place instead of where it is. I should think myself they are going to show that animal; it's uncommon, that stid, more so to see a man riding on it— —. You don't often see a man riding on a

stid." When I asked, "What is a stid?" he replied, "It's something the same family as a cow."

Lieutenant M. (No. 10), an able officer, had been an accountant before joining the Army. I showed him a picture which alluded to the putting forward of the clocks in consequence of the Daylight Saving Act. Standing beside a clock, whose hands point to two, the bride of a day says to her bridegroom, "Look, it's 2 o'clock, and yesterday at this time it was 1," and he replied, "Yes, darling; and yesterday at this time we were two and now we are one!" The patient remarked, "I've got the drift of that. It's not much of a joke. They've stupidly been married; the bridegroom has something stupid to say about yesterday, we were one and now we are two, the same old stupid thing about yesterday, we were two and now we are one — — I could have told the time there better than when you were trying me — —2 o'clock." Q. "What happened at two o'clock just a fortnight ago?" A. "Oh! I see, Daylight Saving Act." Q. "What happened to the clocks?" A. "They were put back an hour. That's a thing I'm rather hazy about. I have to think of that — —forward — — back — —. No, I have to give that up; I've been trying to think that out, and I haven't got to a conclusion yet somehow."

In consequence of this difficulty in appreciating either the full significance of pictures or of printed matter, most jokes become incomprehensible. Suppose, however, the humour consists solely in the detail of the drawing, it is appreciated; but any demand for co-ordination between its various parts or with the text beneath it, meets with little or no response.

These patients cannot play games such as chess, draughts or cards. Nor can they put together "jig-saw" puzzles; No. 8 complained, "I can see the bits, but I cannot see any relation between the bits; I could not get the general idea." This is revealed in another interesting way; he was unable to play billiards because he had no idea how to play off the cushion. He could hit the ball directly, but could not make an indirect cannon.

This want of ability to relate things to one another came out when testing memory for the position of objects in the room or on the table. The patient had no difficulty with his eyes closed in pointing to the position of the window, fireplace, washhandstand, chest of drawers, door, and other pieces of furniture. But asked to say how the washhandstand stood in relation to the fireplace, or the latter to the door, he entirely failed to do so; allowed, however, to say, "The fire is there

and the door there," he pointed with complete accuracy. He knows exactly where they are; he is certain that he can "see them" in his "mind," but he cannot express their relative position.

We can only understand the peculiar affection of writing and reading produced by this disorder of symbolic thinking if we bear in mind that the fault is essentially a want of appreciation of relative significance and intention. At first, as in the case of No. 10, the patient may find it impossible to write the name of an object, although he had no other difficulty in nomenclature; but even at this stage he not uncommonly succeeded in writing the first letter of the word he was seeking. At this time he showed a tendency to use the same symbol "$\mathcal{Q}$" for the letters D, F, O, R, T and V. Five months later each name was correctly given, but he wrote at a tremendous pace as if in a desperate hurry. The words were carelessly written; cylinder became "cilande" and "cylander," ovoid "ovoad," and cube was shortened to "cub." This excessive rapidity was also evident during the man, cat and dog tests, when he wrote from dictation or from pictures. He said, "I write very hurriedly so as to keep track of it." But if he was given the printed card and asked to copy it, he wrote with much greater deliberation and his handwriting improved considerably.

One form assumed by the errors that occur in this group of tests is the tendency to substitute one word for another; "man" is written for dog, "cat" for man, &c., and the order is not uncommonly reversed. This is evidently due to inconstant appreciation, although the task set was childishly easy. One of these patients asked if he might leave out the articles and conjunctions because he could then "do it better" if he wrote "cat, man" only.

Spontaneous writing, such as a letter to friends or relatives, is coherent and reasonable; but these patients complain, "I cannot pull it together very well yet," or, "If I'm writing I am apt to write on and then to wonder if I've put two l's or one; I'm not sure if I've written it wrongly or not." They have, however, a remarkable memory of the general contents; for instance, when No. 10 wrote a test letter to me nearly a year and a half after he had first come under my care, he began: "I have written you three letters, one about flouwers, one, a while later on, on same subject, and a third while I was at Northampton trying to tell you of my progress." This was entirely accurate; and yet he possessed no record by which he could have refreshed his memory.

These patients have no difficulty in reading aloud; when, however, they read silently, they are liable to miss the general sense of the

passage, and to omit words or phrases essential to the argument. This tendency becomes particularly obvious if they are asked to write an account of what they have just read to themselves. For instance, I handed No. 5 his own story in print: "My name is Charles Hewitt and I live at Laurel Cottage, Pilley. Before I joined the Army I worked for Miss Drummond for nearly two years. I worked in the garden and looked after the pony. He was a forest pony, bay, with a dark mane and tail." This he read to himself and wrote, "I was worked for Miss Drummond and looked after a pony and Trap"; then he said, "That's all I remember." He was then asked to read it aloud, which he did perfectly and without hesitation; but all he could communicate in writing was as follows: "I worked for Miss Drummond and I looked after a pony and trap and helped in the garden." This loss of capacity was not based on any defect in the power of writing, for he copied his own story perfectly in cursive handwriting and was able to write a coherent letter to his relatives, mentioning the friends who had visited him by name. Here again symbols could be better utilized for spontaneous thought than for an intellectual effort made to command.

This is not due to want of education, for Lieutenant M. (No. 10) showed exactly the same disability. He read to himself the following passage from the newspaper: "News has been received from the Piraeus that the Greek Government proposes to proclaim martial law throughout Greece, and arrest M. Venizelos and his principal political friends." He at once wrote, "News received from Biraeus, that the Greek Government intend to proclaim martyal law throughon grace." He then read the paragraph aloud correctly, and asked to say what it contained replied, "News has been received from — — from — — that the Government of Greece intends to proclaim martial law throughout the country — — something wrong there — — because the political friends of Venizelos somewhere — —." His account died away in an unfinished sentence.

Drawing, even from a model, shows this want of consecutive memory and intention. These patients do not, as a rule, block out the drawing but tend to begin at some one point and follow round the outline of the object; this is also evident when they attempt to reproduce it from memory. Told to draw an elephant, No. 8 succeeded in producing a four-legged creature with no trunk or ears; but No. 10 and No. 5 could not form a coherent figure of any kind.

None of these patients could draw a plan of a room with which they were familiar. No. 8, who was an excellent draftsman before the

injury, started well, but forgot the windows and the doors; moreover, he placed his seat alongside the fireplace, whereas it was in the middle of the room. He forgot the table in front of him but filled in several details, such as my weighing machine and typewriter, of little comparative importance.

The act of counting was perfect, but simple arithmetical operations were a trouble to all of them. They were subject to curious lapses, such as 6 + 8 = 10 in a simple addition sum, otherwise correctly carried out; sometimes an integer was carried over, sometimes it was forgotten. No. 10, who had been an accountant, could neither add nor subtract; he said, "I seem to get tangled up in the process." In every case coins were named correctly, but there was profound difficulty in stating the relation of any two of them to one another; for example, when a sixpence and a halfcrown were laid on the table, No. 10 said, "Let me see — — twenty-two — — I thought of twenty-four, but it's not"; and yet he gave many of the relative values correctly.

This tendency to confusion comes out perpetually in the operations of daily life. For instance, No. 8 complained, "When I'm going to shave I can't collect my things. I have to look hard at them all and then I am sure to miss some of them. In the same way I have to look at the things on the breakfast table. I see them all but I don't 'spot' them. When I want the salt or the pepper or a spoon I suddenly tumble to its presence. After my belt had been cleaned, the runners had gone back and I could not for the life of me think how to bring them into place." Patients belonging to the semantic group are much troubled by the movement of the streets, which puzzles and bothers them.

§ 5.—*Differences between these Various Forms of Disordered Speech contrasted.*

It has been universally recognized that the clinical manifestations differ greatly in individual cases of aphasia, and such differences have been attributed to a multitude of causes. I have attempted to show that they are produced by dissociation of a definite mental process, which I have called symbolic thinking and expression. They are not due to a loss of motor or sensory power, to destruction of images or to a diminution of general intellectual capacity, but are caused by the breaking up of one aspect of psychical activity analogous, on a higher level, to the sensory dissociations, which may follow a lesion of the post-central cortex. Certain physiological processes necessary for the normal

exercise of the functions of language are disturbed by organic destruction of the brain. At first, in consequence of the widespread effects of this injury, the patient may be unable to speak, to read or to write. But as this state passes away all aspects of the disordered function may not be equally disturbed ; some actions are more easily performed and certain tests are carried out normally, whilst others are grossly affected. Such dissociation of symbolic thinking and expression is responsible for the clinical forms assumed by aphasia.

To each of these clinical forms I have given a name chosen to indicate its characteristic verbal defects. But it must be remembered that, although the power of using words shows the most extensive and gravest disturbance, other actions are affected which have nothing directly to do with such symbols. The name applied to each group of aphasic disorders is drawn from a grammatical source, because it is in the use of language that the changes are most evident and characteristic ; but the functions which suffer extend beyond the limits of verbalization.

Moreover it is well to remember that in each of these pathological groups both word formation and also the correct production of the phrase are affected. Thus in verbal aphasia words are evoked with difficulty and tend to be abnormal in structure. Nominal aphasia leads to defective use and understanding of words as names or indicators. Loss of syntactical power disturbs the internal balance of a word as an orderly rhythmic expression and so leads to jargon ; whilst semantic disorders interfere with capacity to comprehend and retain the general significance of a word as part of a complete act of language.

In the same way the orderly structure or intention of the phrase is affected in one way or another, whatever form the disturbance of symbolic thinking may assume. In verbal aphasia words cannot be evoked at will and retained so as to form parts of a perfectly constructed sentence. When the power of naming is affected, the nominal portions of the phrase are either absent or defective and may be replaced by gestures. But the structure of the phrase suffers most obviously with syntactical disorders. The normal rhythm of the sentence is destroyed, speech becomes syncopated, and those words which serve to bind together its constituent elements are absent or ineffective. Lastly, the patient who suffers from a semantic affection of speech pauses in his conversation, like a man who has lost the thread of what he wanted to say ; he falls back on expressions such as, "Well, you see it's like this," and sentences may die out unfinished.

All these disorders have both a formative and emissive aspect. In verbal aphasia the latter is the more prominent and obvious, but

it must not be forgotten that there is also another side; these patients cannot retain with certainty a series of words or some unusual expression and this makes it difficult for them to read to themselves with ease and complete understanding. Moreover, the words uttered internally may be badly formed and so lead to confusion; No. 17 complained, "I can't read a book because I'm bothered, when I say the words to myself"; this comes out particularly clearly on attempting to write to dictation. With nominal aphasia the disturbance is evident both on attempting to name an object, and to carry out a command given orally or in print. Syntactical aphasia consists of a gross disorder of the emissive side of speech; but there is also difficulty in executing orders communicated by word of mouth. Spoken language is not perfectly understood, although comprehension of print may be undisturbed. In semantic affections the predominant feature consists in failure to receive a uniformly correct impression of the general significance of words or phrases; on the emissive side the defect appears as a loss of executive intention. This is particularly evident in drawing and in games such as billiards.

It must not be supposed that the various aspects of symbolic thinking and expression usually appear in these isolated forms. Most cases of aphasia, especially in the earlier stages, show evidence of widespread disorders of language. Many of these changes disappear with the passing away of neural shock; but the loss of function becomes confined to one of these groups in a few cases only. On the following tables I have put together those instances where the disturbance seemed to correspond most nearly to a dissociated affection of one aspect of symbolic thinking and expression, and I have attempted to show how the patients behaved to some of the tests. It is impossible on a general table to give an exact account of the nature of the responses, but the diagrams may help to make clearer the contrasting features of the different varieties of disordered speech. In all cases where the series of answers was in any way abnormal some indication is given in words of the form they assumed. Where the space is filled by a O the act was performed perfectly.

When aphasia is looked at from this point of view, it is obvious that no single test can be expected to reveal one form only. All the methods of examination I have described were designed to show how the patient behaves when a certain task is set before him in different ways. But, when the results obtained with such tests, in the various cases, are put together, certain conclusions emerge which can be summarized as follows:—

TABLE VII.—ARTICULATORY SPEECH.

	Word formation	Intonation and stress	Syntax	Repetition of words said by observer
Verbal.				
No. 4 ..	Great difficulty ..	Syncopated and broken	O	Good
No. 6 ..	Profound loss; at first dumb	Syncopated and broken	O	Good
No. 17 ..	Great difficulty ..	Syncopated and broken	O	Good
Nominal.				
No. 2 ..	Good except when doubtful of a name	O	O	Words perfect; content inaccurate
No. 7 ..	Extreme difficulty of expression	O	O	Slow; tends to change content
Syntactical.				
No. 14 ..	Jargon	Hurried and jerky	Grossly affected	Impossible
No. 15 ..	Jargon	Hurried and jerky	Grossly affected	Very bad; jargon
No. 13 ..	Jargon	Hurried and jerky	Grossly affected	Jargon
Semantic.				
No. 10 ..	O	O	O	O
No. 5 ..	O	O	O	O
No. 8 ..	O	O	O	O

TABLE VIII.—COMMON OBJECTS CHOSEN AND NAMED.

	Choosing object similar to one shown	Choosing object named aloud	Choosing object named in print	Naming object indicated	Writing name of object indicated	Duplicate placed in normal hand
Verbal.						
No. 4 ..	O	O	O	Slow; articulation defective	Spelling bad..	O
No. 6 ..	O	O	O	Slow	Slow	O
No. 17 ..	O	O	O	Slow; articulation defective	Writing and spelling bad	O
Nominal.						
No. 2 ..	O	Slow	Slow	Gross loss ..	Gross loss ..	O
No. 7 ..	O	Slow	Defective	Gross loss ..	Impossible ..	O
Syntactical.						
No. 14 ..	O	O	O	Incomprehensible jargon	Affected ..	O
No. 15 ..	O	O	O	Tendency to jargon; sense correct	—	O
No. 13 ..	O	O	O	O	O	O
Semantic.						
No. 10 ..	O	O	O	O	Grossly affected	O
No. 5 ..	O	O	O	O	Slow; badly spelt	O
No. 8 ..	O	O	O	O	O	O

TABLE IX.—CLOCK TESTS.

	Imitation	Oral command	Printed command	Telling time	Writing time
Verbal.					
No. 4 ..	O	O	O	Content correct; articulation bad	O
No. 6 ..	O	O	O	Same ..	O
No. 17 ..	O	O	O	Same ..	O
Nominal.					
No. 2 ..	O	Gross loss ..	Gross loss ..	Gross loss ..	Gross loss
No. 7 ..	O	Gross loss ..	Gross loss ..	Impossible ..	Gross loss
Syntactical.					
No. 14 ..	O	Definitely affected	O	Impossible ..	Correct in sense, extraordinary nomenclature
No. 15 ..	O	Slow; hesitating	O	Slow; hesitating	Same
No. 13 ..	O	O	Almost perfect	Slightly affected	O
Semantic.					
No. 10 ..	O	Gross confusion	Gross confusion	Some loss (later perfect)	Definite loss; confuses hands
No. 5 ..	O	Gross confusion	Confusion ..	O	Confused
No. 8 ..	Somewhat confused	Confused ..	Gross confusion	O	O

TABLE X.—HAND, EYE, AND EAR TESTS.

	Imitation, face to face	Imitation in mirror	Pictorial command	Pictorial command in mirror	Oral command	Printed command
Verbal.						
No. 4 ..	Affected ..	O	Affected ..	O	O	O
No. 6 ..	Affected ..	O	Affected ..	O	O	—
No. 17 ..	Slightly affected	O	Slightly affected	O	O	O
Nominal.						
No. 2 ..	Grossly affected	Not quite perfect	Grossly affected	Affected ..	Grossly affected	Grossly affected
No. 7 ..	Grossly affected	Very slightly affected	Grossly affected	Affected ..	Grossly affected	Grossly affected
Syntactical.						
No. 14 ..	Slow, but correct	O	Slow, but correct	O	Affected ..	O
No. 15 ..	Slow, but correct	O	Slow, defective	O	Affected ..	Affected
No. 13 ..	Affected ..	O	Affected ..	O	O	O
Semantic.						
No. 10 ..	Gross loss ..	Affected ..	Gross loss	Gross loss	Gross loss	Gross loss
No. 5 ..	Gross loss ..	Slightly affected	Gross loss	Affected ..	Affected ..	O
No. 8 ..	Affected ..	Slightly affected	Gross loss	Affected ..	O	O

(*a*) *Verbal aphasia.*—In severe forms of this disorder the patient's utterances may be reduced to "Yes" or "No," and even the words cannot always be evoked for voluntary use. As speech returns his vocabulary increases, his enunciation is slow and halting. Any word he is able to recall can, however, be used for naming an object; it may be so badly pronounced as to be scarcely recognizable, but it is applied correctly. When the patient attempts to repeat what has been said to him the articulatory sounds are imperfect, but he can usually utter more words than are possible spontaneously. It is characteristic of this form of aphasia that words are evoked with difficulty and tend to be abnormal in structure.

After the stage of neural shock has passed away, the power of choosing an object to oral or printed commands becomes perfect; for in this case the words he requires are presented to the patient by the observer. Even orders necessitating choice, such as the hand, eye and ear tests, can be carried out correctly if given in print or in words spoken aloud.

At first writing may be extremely difficult, or almost impossible. But as his spoken vocabulary increases the power of writing is regained, although to the last it tends to show the same errors as articulatory speech. These patients cannot spell, and find difficulty in remembering the order of the letters even in simple words. They write more easily to dictation, but are unable to carry in the memory a string of words or a long phrase. They can copy with ease printed matter in cursive handwriting.

The power of reading to themselves with enjoyment is spoilt by the difficulty in remembering a series of words accurately; they are frequently compelled to look back to the beginning of a long sentence in order to obtain its full meaning.

The verbal aspect of numerals is affected, but not their significance. Thus, when looking up the page of a book or scoring at cards the patient may utter the wrong number, but acts as if he had said the right one. Simple arithmetical operations can be carried out correctly, except in very severe cases; then it is not the process of addition or subtraction which is forgotten, but the act fails because of difficulty in remembering the requisite figures.

These patients can draw, play card games and enjoy jokes set out in print or in pictures. In fact, the disorder from which they suffer affects mainly verbal structure and words as integral parts of a phrase; their nominal value and significance are perfect.

(b) Nominal aphasia.—From the verbal point of view this is essentially a defective use of names. Not only does the patient fail in naming objects placed before him, but he has difficulty in employing expressions which give to words their value as a distinctive nominal indicator. For example, the only difference between "five minutes to eight" and "five minutes past eight" lies in the prepositions "to" and "past," and it is here that the patient is liable to fall into error rather than over the numbers five and eight.

When asked to point to an object named aloud or in print, the choice, if correct, is made slowly and with difficulty; for it is the nominal value of the word which is affected and not, as in verbal aphasia, its structural formation. This comes out strongly when the words are made to carry a command requiring choice, and the hand, eye and ear tests are badly executed to verbal orders given aloud or in print.

This inability to find correct names applies to the letters of the alphabet. The patient may be able to utter them in sequence to a varying extent, but he cannot be sure of naming isolated letters correctly. This greatly hampers his power of reading; for, when arrested by a word, he attempts to spell it letter by letter and is confused by the false names he is liable to give them.

Writing is gravely affected, and, although the patient can copy printed matter correctly in capitals, he may be unable to do so in cursive handwriting.

Repetition *viva voce* is perfect provided nothing further depends on the act; but writing to dictation, or any other action demanding choice, is performed with difficulty to spoken commands.

These patients can usually count, but suffer from defective appreciation of the value of single numbers. Thus No. 2 did not fully recognize the significance of such a number as 18 until he had counted up to it. This want of immediate comprehension of the nominal meaning of figures leads to want of ability to carry out simple arithmetical operations. This defect varies greatly in form and extent, but it was present to an obvious degree in my most highly educated patient (No. 2).

Games, such as cards, which demand rapid and correct recognition of names and the power to register a score, are impossible. No. 2 was, however, excellent at chess and could put together a puzzle without difficulty, whilst other patients of this group played draughts and dominoes with pleasure.

They could draw from a model or from memory after it was removed from sight; but, when asked to draw some such figure as that of an

elephant, the result was extremely unsatisfactory. All the distinctive parts of the beast were usually omitted ; and yet No. 2 made spontaneously excellent drawings of the animals he had shot before his injury.

These patients had no difficulty in appreciating the full significance of pictures and could understand jokes provided they were not conveyed in long and complicated phrases.

One of the most instructive forms assumed by this loss of function is the difficulty experienced by the patient in drawing a ground plan of some room with which he is familiar. However badly the verbal aphasic may draw, he succeeds in indicating the relative position of the windows, doors and principal pieces of furniture. But the patient with nominal aphasia fails to produce a correct plan, and usually slips away with an attempt to express the constituent parts in elevation ; and yet if he is asked "Where is the table ?" or "Where is the window ?" he can usually point to their situation. A similar want of power to indicate relative positions appeared during the tests for naming common objects or colours. They were laid on the table in front of the patient and he soon became familiar with the position of each one of them. They were then hidden from his sight, and when a duplicate was shown to him he usually had no difficulty in pointing to the situation of the similar object, although he could no longer see it. But if the name only was given to him, either aloud or in print, he had difficulty in pointing to the relative position of the object on the table ; for, given the word, he attempted to remember the names of the test objects in their order and became, in consequence, confused.

(c) *Syntactical aphasia.*—This is an easy form to distinguish because the patient talks jargon. The verbal aphasic speaks slowly and with difficulty ; each word is produced with an effort. But with uncomplicated syntactical defects, speech is voluble and words are emitted with great rapidity. Sometimes each one is comprehensible, however difficult it may be to gather the full meaning of the phrase ; but in other cases the words uttered are pure jargon. One of the most characteristic features of this form of speech is the want of grammatical coherence ; the words tend to be ejected in a disconnected stream.

The power of naming objects placed before him may be retained by the patient in spite of the jargon by which he is hampered. Not infrequently, when he cannot utter a word or when the sound emitted is incomprehensible to his auditor, he can write the name correctly. In the lighter forms of this affection he may vary greatly in the expressions he uses for the same object, though each of them bears some distinct

relation to its essential nature. Thus a pencil may be called at one time "black-lead" at another "blacking."

These patients can understand what they read to themselves provided they do not attempt to formulate it in words. Thus No. 14 read the daily papers and could demonstrate to me correctly the progress of the War by pointing to large scale military maps; but as soon as he tried to describe some event in words he became confused.

The difficulties experienced in writing show that internal speech is also disturbed by jargon. Single words, especially names in common use, can be written correctly, but any attempt to convey a formulated statement is liable to end in confusion. Patients suffering from the more severe degrees of this affection cannot write a letter, but in the lighter cases writing is easier than speech. They can all copy correctly and transcribe printed matter into cursive handwriting.

This disorder is essentially one of auditory balance and rhythm. Syntax, as the expression of this aspect of phrase formation, suffers greatly and, combined with the destruction of the internal balance of words, leads to jargon.

(*d*) *Semantic aphasia*.—So far the names I have applied to the various forms of disordered speech have borne some relation to the verbal defects. For this group, however, it is difficult to find a suitable designation that will express the essential nature of the disturbance, which extends beyond the limits of organized words. I have chosen the term "semantic" as a label for this form of aphasia because the affection comprises a want of recognition of the full significance or intention of words and phrases. But other functions suffer that have nothing to do directly with verbalization. The patient may be unable to appreciate the complete meaning of a picture although he recognizes all its details. He can carry out a manœuvre, where each action suggests the next, but is unable to do so if he is compelled to formulate it as a whole. Power to bear in mind the ultimate intention of the derived action is diminished and the patient has no firm recognition of the final goal of his efforts.

He has no difficulty in forming words and can repeat what is said to him. But if he is told some simple story and asked to reproduce it aloud or in writing, many of the essential elements are omitted; this also occurs to an even greater degree after he has read it to himself silently. He cannot retain that total conception of episodic sequence which is necessary for complete narration.

The clock-tests reveal the nature of this disorder in a striking

manner. The patient confuses the two hands, does not know how to approach the task of setting them to verbal or printed command, and forgets the meaning of "past" and "to" the hour. Even the attempt to imitate directly on one clock the time set on another may lead to confusion and be carried out uncertainly. For whatever the test the patient is liable to misunderstand the intention of what he is asked to do. On the other hand, except in the gravest cases, he has little difficulty in telling the time provided he is allowed to keep the clock in front of him until he has given his answer.

Arithmetical operations become impossible or are carried out uncertainly and with difficulty. The patient may be unable to add or subtract because the mathematical process itself is incomprehensible.

He fails entirely to comprehend most jokes, especially if they demand the complete understanding of a picture and its printed legend. He cannot play cards games or put together puzzles, which confuse him greatly.

These semantic disorders interfere seriously with the actions of daily life and render the patient useless for any but the simplest employment; and yet his memory and intelligence may remain on a comparatively high general level. He does not forget people or places and can recall accurately events both recent and remote. Thus No. 10 was able spontaneously to remember the subject of the three separate letters he had written to me at various sittings during three years. But had I set him the task of repeating accurately a simple story, which he had been told or had read to himself, he would have become confused and many factors of importance would have been omitted.

Chapter V.—Symbolic Thinking and Expression.

Now that I have described the various dissociated forms assumed by disorders of speech in consequence of a unilateral lesion of the brain, it is possible to consider in detail the general nature of the functions which are affected. These I have grouped together under the title "Symbolic Thinking and Expression"; but I am anxious that this term should not be considered as defining the limits of the disturbance. I should have preferred to adopt some entirely neutral appellation, and to define its meaning by enumerating seriatim the various activities which are found to be affected.

To each of the partial forms of aphasia described in this paper a name has been given, indicating the characteristic disorder of speech,

although the use of words is by no means the only function affected. These terms are not intended to define the limits of the disturbance, but serve simply as a memoria technica. In the same way I have combined the functions which are affected by a unilateral lesion of the brain under the general heading of "Symbolic Thinking and Expression," because, in the majority of instances, the gravest disturbance is shown in the use of such symbols as words and figures. Any mental activity is liable to suffer which demands the perfect reproduction of some symbol between its initiation and fulfilment. I do not, however, believe that it is possible to include within one categorical definition all the activities which experience shows to be affected; and yet from a physiological point of view they form a group as definite as those which underlie sensation.

Since it is impossible to define the exact limits of what I have called "symbolic thinking and expression," I shall enumerate the various actions comprised under this heading, which are affected by unilateral lesions of the brain. For the present I wish to exclude cases of bilateral affections, and all patients who showed evidence of definite destruction of sensory images. These must form the subject of an independent research; but they cannot be understood until the principles laid down in this paper have been clearly grasped.

It is not the "general intellectual capacity" which is disturbed by these lesions of the brain, but the mechanism by which certain aspects of mental activity are brought into play. Behaviour is affected in a specific manner; an action can be carried out in one way, but not in another. Thus, all these patients could choose from amongst a series of shapes or colours, the one that had been shown to them. They had no difficulty in picking out an object, which corresponded to a duplicate placed in the hand out of sight. All these are acts of direct matching; but the more definitely the task demands for its perfect execution symbolic formulation, the more certainly will it be badly executed.

The highest stage is reached in formulating a proposition, and, as Jackson pointed out, it is this form of mental expression which suffers most severely. Many patients who can choose an object correctly to oral or printed commands, can neither evoke the same spontaneously nor read it aloud; for both these actions require symbolic representation. If the word is given, it can be matched with some object on the table, but the adequate symbol cannot be called up at will.

When, however, the same patient is compelled not only to evoke a

set of symbols, but to use them as a preliminary to action, as in the hand, eye and ear tests, he may be unable to execute oral or printed commands. For under such circumstances he must first formulate the proposition and then carry it into action correctly.

Any modification of the task, which lessens the necessity for symbolic representation, will render its performance easier. Thus a patient who finds extreme difficulty in imitating gestures, when sitting face to face, makes no mistakes when they are reflected in a mirror; for this is not a propositional act but one of direct imitation. If, however, he is asked to write down the movements visible in the mirror, he falls into the grossest errors because he is now obliged to formulate in verbal symbols what he sees.

The higher the propositional value of the mental act, the greater difficulty will it present. Thus a patient may execute a printed command to hold up his hand, although he is unable to carry out an order to touch with it his eye or his ear. The addition of the second factor has rendered the task too difficult; in the one case he appears to be able to read, whilst in the other he seems to suffer from "alexia." The larger the number of possible alternatives presented by the order, the more certainly will the desired action be defective. Thus it is possible by means of a series of printed cards to grade the severity of the patient's disability. He is first required to carry out a series of movements such as "lift your hand," "shut your eyes," &c., which name one part of the body only. Then the right or left hand is specified, and the task is gradually increased in severity until at last it is brought up to the multiple alternatives of the full hand, eye and ear tests. Somewhere on this ascending scale of difficulty the aphasic will break down, and this gives a rough indication of the extent of the loss of ability to carry out printed commands.

On this principle we can explain the behaviour of No. 2, who was unable to name colours when asked to do so, but never failed to describe them appropriately if he was allowed to use a simile or metaphorical phrase. For example, black was "what you do for the dead"; finally, during later observations at this stage, he was able to shorten this phrase to "dead." He could now name the colour by a single word "dead," because of its metaphorical significance, although he was unable to call up the more directly nominal expression "black."

The large majority of aphasic patients can copy printed matter into capital letters; this is purely imitative. But to copy it in cursive handwriting is an act of transliteration, demanding a certain degree of

symbolic thinking. It is not surprising, therefore, to find that this form of activity is disturbed in some cases of aphasia.

It is comparatively easy to say the alphabet, the days of the week or the months of the year in sequence, provided it is possible to form the necessary words; long familiarity has made such tasks almost automatic. But to recognize immediately the significance of any single letter, day or month demands a higher order of symbolic thinking. Thus, a patient who can say such a series correctly may be unable to spell out the letters of a word or to comprehend the meaning of a definite date such as "Tuesday, the twentieth of March." He can set the clock at "4.45," but not when he is told to place the hands at "a quarter to 5." For in the first instance he places the short hand at 4, and then swings round the other consecutively up to forty-five minutes; in the latter he sets the hour hand at 5, and then stops, puzzled by doubt as to the nominal significance of the words "a quarter to."

The clock-tests also reveal the existence of symbols, which are neither expressed words nor numbers. The short and long hands have acquired a significance which converts each one of them into a direct symbol, and they are confused or used wrongly in many forms of aphasia (Table IX, p. 152). Moreover, we are in the habit of dividing up the space between any two numbers on the clock face into portions of an hour. If we are told to set half-past one, we not only bring the long hand opposite to the figure 6, but we bisect the space between 1 and 2, and place the hour hand in this position. The space between the two figures marking the hours has in itself a symbolic value. In many cases of aphasia this is affected, and the patient no longer sets the hour hand at a point proportionate to the position of the minute hand; he places the former opposite the figure 1, whilst the latter points to 6. Or, more confusing still, when told to set "a quarter to six," he may place the short hand at 6 and the long hand at 9, so that it is impossible to discover without questioning him whether he intended to set 6.45 or 5.45. Thus, not only the hands of the clock, but their relative position on the face have acquired a symbolic value, which is disturbed in many cases of aphasia.

The activity of the cerebral cortex is particularly associated with determination of the relations between external objects. Man developed the power of speech at a time when he had already acquired discriminative powers of a high order. The line along which the higher cerebral centres progressed was apparent in an increased

capacity to distinguish variations of intensity, similarity, and difference, and spacial relationships. To this speech was added, which, apart from its emotional aspects, is concerned with expressing these relations. Definite symbols such as words and numbers were invented to register these attributes; but they are not the only relative factors, which may be disturbed in disorders of symbolic thinking and expression. So long as the patient has to choose an object from those in front of him, which corresponds to a duplicate shown to him or placed in his hand, he can remember the order in which they lie on the table even when they are hidden from his sight. But as soon as he is asked to formulate their order, that is, their relation to one another, he fails to do so. In the same way he may be unable to draw a plan of some room with which he is familiar, although he can recall and indicate correctly the situation of each single object.

In the same way No. 10 remembered that when buying tobacco he placed two shillings on the counter and received two ounces and threepence change; but he was unable to say how much it cost. He could register the facts correctly, but could not relate them to one another.

In the same way sense impressions and images can be appreciated and recalled, but the aphasic may have difficulty in expressing the relation between them. Thus, when tested with the compass-points, he may be entirely unable to answer correctly in speech or writing although he possesses the necessary words; and yet there is no reason to suppose that sensibility is affected. For if the figures 1 and 2 are written on a sheet of paper, he can indicate correctly whether he has been touched with one or two points. Under the ordinary conditions he is compelled to formulate his sensory impressions and to express their relation to some other condition. But when the test is carried out according to the second method, he has only to match his sensory image with one of two patterns in front of him.

Another form of activity, which is particularly disturbed in the semantic group of speech defects, is what may be called the ultimate intention of the symbol. These patients have no clear or certain conception of the goal of the action they are asked to perform. This is profoundly evident in all that concerns words and figures. But it is not the individual words in their primary sense which are gravely affected. No. 10 understood the words "summer" and "time," and also was well aware that "summer time" signified that the clocks were changed with the advent of summer. But he was entirely unable to say whether they were put forward or back, and tried in vain to work

out the problem. In the same way these patients cannot add or subtract with certainty, because it is the processes of arithmetic which have been lost, and not the direct significance of figures.

This failure to formulate the intention or ultimate goal of a desired action leads to loss of capacity to perform tasks not directly associated with words or figures. Thus, when No. 10 was threading a quadrilateral frame for his bee-hives, he could carry out the operation if the action consisted in bringing the wire across from one side to another, and then back again through neighbouring holes; but as soon as he attempted to go from corner to corner he failed entirely. He could carry out a continuous action, but fell into difficulty when he was compelled by the discontinuity of the task to formulate his intention. In the same way a young officer, No. 8, was unable to put together his belt when the slides had been displaced.

In this chapter I have attempted shortly to bring together the various activities which are disturbed, when symbolic thinking and expression are affected. I believe that they form a group of mental processes, which can be defined at present by enumeration only. Some may object to the name I have selected; but this has been chosen solely because the disorder of these functions is most often and most profoundly manifested in the use of words and figures.

Summary.

(1) The results set forth in this paper are based mainly on the investigation of young men suffering from gunshot wounds in various portions of the head (p. 90).

(2) Certain new serial methods of examination have been adopted; these make it possible to utilize the inconstant answers, characteristic of all cortical lesions, which are so confusing unless the examination is made in a systematic manner (pp. 89-107).

(3) These observations show that disorders in the use of language, due to an unilateral lesion of the brain, cannot be classed under the categories of isolated affections of speaking, reading or writing (pp. 108-111).

(4) They cannot be explained as due to destruction of images, "visual," "auditory" or "motor" (pp. 111-113).

On the other hand, "word-blindness," "mind-blindness," and what Jackson called "imperception," are all associated with more or less disturbance of the power to form images, complicated in some

cases by those affections of language which form the subject of this paper.

(5) The "motor" aspect of these disorders of language is not solely an "anarthria" or high-grade articulatory defect. Careful examination shows that, in cases of so-called "motor" aphasia, not only external speech, but certain aspects of internal verbalization are affected. Thus the patient may find considerable difficulty in imitating movements made by the observer sitting face to face or in carrying out the same actions to pictorial command; and yet they may be perfectly executed, when reflected in a mirror. For in the first case some formulation in words is required, whilst in its second form the test is verbally an act of uncomplicated imitation. Moreover, writing tends to show faults in word formation of the same order as those of articulatory speech and spelling may be affected (pp. 113-115).

(6) I have grouped together the functions, which are affected in aphasia and kindred disorders of speech, under the heading of "symbolic thinking and expression." This name has been chosen because the gravest and most definite disturbance is to be found in the use of words, figures, and other symbols (pp. 115-119).

(7) But this term must not be supposed to define exactly the limits and extent of the actual loss of function, which can be discovered by examination. For not only may the power of using words and figures in speaking, reading and writing be affected, but there are other tasks which the patient cannot execute with certainty and correctness. He may be unable to formulate or draw a plan of the relative position of objects with which he is familiar, although he can indicate the site of each one of them individually. He mistakes the significance of the two hands of the clock and fails to recognize the proportionate value of the space between the figures of the hours. He can draw from a model, but may be unable to reproduce the form of an elephant to command. He fails to comprehend the full significance of a picture, although he recognizes the details of which it is composed. Any act is liable to suffer which requires for its perfect performance the antecedent formulation of the ultimate intention or goal towards which it is directed (pp. 157-162).

(8) The more nearly a symbolic action approximates to a proposition the greater difficulty will it present and the patient will probably fail to execute it correctly. The closer the task corresponds to matching two sensory patterns the less likely is it to be affected by these disorders of language. Highly complex symbolic acts suffer more gravely than those of a lower propositional value (p. 159).

(9) Under the influence of lesions situated in different parts of the brain the various functions comprised under "symbolic thinking and expression" may become dissociated. This is analogous to the effects produced on sensation by injuries to the cerebral cortex. Each of these dissociated forms represents a fraction of the complex and highly developed psychical process (pp. 148-157).

In most cases of aphasia two or more of these aspects of symbolic formulation are affected; but in order to comprehend the nature of the disordered functions it is necessary to select for examination patients in whom the disturbance is as nearly as possible confined to one of these forms of dissociation. Subsequently it is possible to understand the more complex aphasias and to enumerate in full the various actions comprised under symbolic thinking and expression.

(10) The various dissociated forms of symbolic thinking and expression may be comprised under the following headings:—

(*a*) Verbal Aphasia. This is essentially a defect of word formation. Words are evoked with difficulty and the vocabulary is greatly restricted. Enunciation is slow and halting. Writing tends to show the same sort of errors as articulatory speech, and spelling is defective. The patient has difficulty in reading to himself with pleasure, because he is unable to retain in his memory a long series of words. Numerals are affected to a slighter degree; their significance may be recognized and acted on correctly although they are wrongly enunciated. As speech returns, commands given in spoken or printed words can be executed; but orders which necessitate the evocation of some word or phrase may be carried out badly. These patients recognize, however, whether the task they are attempting has been performed correctly or not. They can draw, play card games, and comprehend jokes set out in print or in pictures (pp. 121-127).

(*b*) Nominal Aphasia. This is essentially a defective use of names and want of comprehension of the nominal value of words or other symbols. The patient reads with extreme difficulty, especially if he attempts to spell out the words. Writing is gravely affected and he may be unable to copy print into cursive handwriting. Writing to dictation and all actions demanding choice are performed with difficulty to spoken commands. Counting is possible to a varying extent, but the significance of numbers, the power to carry out simple arithmetical operations and appreciation of the relative value of money are usually more or less affected. The power to draw a strict ground plan of some familiar room is defective. These patients cannot play cards but chess and draughts may be possible (pp. 127-136).

(*c*) Syntactical Aphasia. This is an easy form to distinguish because the patient tends to talk jargon. Not only is articulation of the word ill-balanced, but the rhythm of the phrase is defective and there is want of grammatical coherence. These patients can read if they are not compelled to reproduce the meaning in words. Writing is usually less affected than external speech, although it tends to be disturbed by verbal jargon (pp. 136-142).

(*d*) Semantic Aphasia. This consists in a want of recognition of the full significance of words and phrases. The patient may understand each word or short phrase, exactly as he can comprehend the details of a picture; but the ultimate meaning escapes him. He fails to comprehend the final aim or goal of an action imposed on him from without. He cannot formulate symbolically a general conception, although he can enumerate the details of which it is composed. He can read and write, but the result tends to be inaccurate and confused. Counting is possible and the value of numerals can be recognized, but appreciation of the nature of arithmetical process is defective. These patients cannot play games, and jokes set out in print or pictures are rarely apprehended in their full significance.

CONGENITAL FAMILIAL SPINAL MUSCULAR ATROPHIES AND THEIR RELATION TO AMYOTONIA CONGENITA.

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SINCE Oppenheim, in 1901, described a disease which he called myatonia congenita (amyotonia congenita), accounts have been published of more than sixty cases of this disease, ten of these with autopsy. Some features of these cases are striking. One is that no case of amyotonia congenita in adults could be found in the literature. Possibly the persons who have had the disease are cured or dead. If the end result has been recovery it would conform well with the theory of Oppenheim and other authorities on the disease, that it is due to a retarded development of the muscles, and that the prognosis of the disease in all cases is good. This hypothesis conforms with Spiller's and Lereboullet-Badouin's findings at autopsies in some cases, where the spinal cord was found to be normal and only slight changes in the muscles were demonstrable. Spiller's case is, however, somewhat doubtful, as it was complicated by ocular phenomena and imbecility. Bing found normal muscle at a biopsy in a case of amyotonia congenita. However, this did not prove that all the muscles were normal. In eight other cases—Rothmann, Reyer-Helmholtz, Archangelsky-Abrikosoff, Marburg, Collier-Holmes, Griffith-Spiller, Laignel-Lavastine-Voisin, and Kaumheimer—considerable changes were found in the spinal cord and the muscles: atrophy of the anterior horn-cells, the anterior roots and the muscle fibres, in some cases also hypertrophic muscular fibres. These findings do not point to a retarded development, but rather to a much more profound change, and it might be supposed that these children, if they had lived, would have been affected by severe and irreparable atrophies and paralyses. Further, the histories of cases of amyotonia congenita show another problem; it is that of the heredo-familial nature of the disease. As is well known, in the overwhelming majority of cases no data pointing to heredity have been found, but in a few cases there has been a heredo-familial history. In the literature I have found four: the cases recorded by Beevor, Silvestri, Sorgente, and Skoog. This relation may only be explained in one of two ways.

One is, that amyotonia congenita is a heredo-familial disease conforming to Mendelian laws, but certain conditions must be present to determine its development. The other and more probable explanation is that the term amyotonia congenita really covers two different diseases, of which the one is heredo-familial, the other is not, a great difference existing between the heredo-familial and the non-familial diseases. The theory that the so-called amyotonia congenita represents two different diseases would also explain the other fact, that many patients are probably cured, while in other cases with an apparently less severe onset death has followed, and microscopy has shown extensive atrophy of muscle and disease of the anterior horn-cells.

If this last theory is correct, there exist two diseases: (1) Amyotonia congenita, which is characterized by a congenital hypotonia of the muscles, hyperflexibility of the joints, and slight pareses or pseudopareses, but no atrophies; this disease is not heredo-familial and the prognosis is comparatively good; only few cases (Spiller's and Lereboullet-Badouin's) have come to autopsy.[1] (2) A heredo-familial congenital muscle-disease, which bears some resemblance to amyotonia congenita, but shows, in addition to the hypotonia and hyperflexibility, marked atrophy of the muscles and is heredo-familial; in a certain number of these cases the heredo-familial factor cannot be directly shown as in the case of all the heredo-familial diseases; this last disease should show atrophy of the anterior horn-cells and of the muscle-fibres.

These theories are not alone supported by the above-mentioned considerations, but primarily by the following small series of cases.

The problem as to the relation between the myopathies and amyotonia congenita has been much discussed in the Neurological Section of the Royal Society of Medicine after lectures by Beevor, Batten, Collier and Homes, Fearnsides and Smith, Whait and Naish. Collier and Batten held antagonistic points of view. Collier did not see much clinical resemblance between amyotonia congenita and myopathy. He considered as the chief points of distinction the following: (1) The absence of any familial tendency. (2) The onset of the disease; it is either congenital or it appears rapidly after an acute illness. (3) The course of the disease is usually towards recovery. (4) The distribution of the muscular affection. (5) The invariable absence of

[1] As for the so-called acute forms of amyotonia congenita, I agree with Spiller and Batten, who consider them to be acute poliomyelitis, which undoubtedly may occur in the first months of life.

any tendency on the part of the disease to spread to regions previously unaffected. (6) The absence of any local muscular weakening or wasting. (7) The condition of the deep reflexes which are absent at first and subsequently return.

Batten inclined rather to consider amyotonia congenita as belonging to the myopathies; he suggested that specially Whait's case showed a connecting link between the so-called amyotonia congenita and the myopathies. Also Head had been struck with the resemblance shown by most of the cases of amyotonia congenita to myopathy.

I would explain these divergences thus. On the whole Collier is right in considering amyotonia congenita and progressive muscular dystrophy as two different diseases with the above-named characteristics. But even the two cases of which Collier gives a pathological-anatomical description were myopathies, not cases of a myotonia congenita. He says about the one case that it is the only one to show an increase of the weakness; in the upper extremities the muscles felt peculiarly soft and homogeneous, and could not be differentiated from the skin and subcutaneous tissues. Pathologically there was a great similarity in the muscle changes in the two conditions; the spinal changes observed in one of his cases have been repeatedly observed in cases of true myopathy.

The subject of atrophies must be considered with some reserve. In many cases the muscles of infants are covered by a considerable layer of fat, so that it is impossible by simple inspection and palpation to judge if there is general or localized muscular wasting. Only the X-rays can, in the case of certain muscles, indicate atrophy.

In published accounts I have found only a very few cases which the author has considered, not as amyotonia congenita, but as congenital progressive muscular dystrophy. Jendrassik mentions a case of congenital muscular dystrophy in which not only the muscles of the extremities, but also the facial and the eye muscles were affected. He mentions two other cases (Winkler and Weide; no reference to literature). Ziehen describes a case, 18 years old, with paralyses of the facial and eye muscles, and of the pectoralis major muscle. This condition was, possibly, congenital. Guthrie, in 1899, showed before the Clinical Society in London a patient whom he described as a case of congenital paralysis due to myopathy. But he said at the discussion that it undoubtedly must be regarded as an instance of amyotonia congenita.

Beevor, who originally considered his cases (also examined by

Batten) as congenital myopathies, declared in the discussion at the Royal Society of Medicine that he now would consider them as cases of amyotonia congenita. Later authors have usually considered Beevor's as cases of amyotonia congenita. Batten published in *Brain*, 1911, eight cases of Werdnig-Hoffmann's type of spinal muscular atrophy.

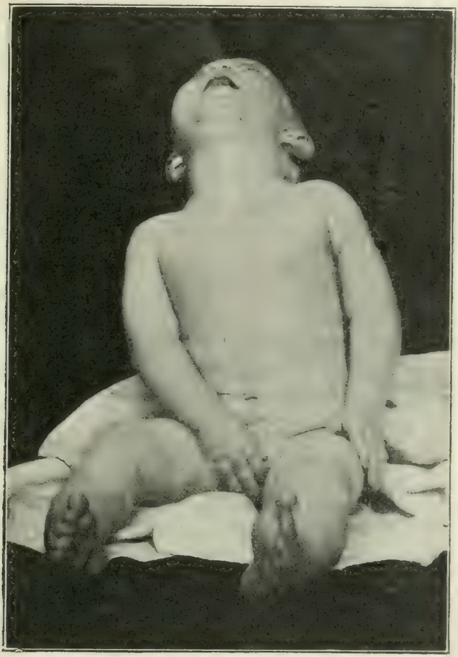

FIG. 1.—Case 1, A. F.

In one of these the paralysis was noticed when the child was two weeks old, in another it was noticed one week after birth; a third case was said always to have been weak. It is not probable that the disease should have occurred acutely so soon after birth; it is more likely that at least two of Batten's cases were congenital. Howard has further published a case of congenital progressive muscular dystrophy (cit. Spiller; the original article was not seen by me).

Wimmer published, in 1905, a case of congenital disease of the muscles in a boy, aged 16 months. There was no similar disease in the family (cf. *infra*). The mother was well during pregnancy, but she hardly ever felt fœtal movements. The child was born in a natural way. Immediately after birth he was flaccid, could not lift his arms or flex them fully at the elbows, and there was practically no movement about the hips. The weakness of the muscles increased, and at the age of 16 months he could not raise himself, and his head fell back unless he sat up absolutely straight. His mental development seemed normal. There was great hyperflexibility about all the joints. Electrical examination showed slow contractions of the proximal muscles. Tendon reflexes absent. Pain reaction normal. Wimmer suggests that the case was scarcely one of amyotonia congenita, but rather was related to Werdnig-Hoffmann's progressive muscular atrophy, in spite of there being no obvious element of heredity. But this appeared later.

Ten years later I was consulted in the nerve-polyclinic of the Rigshospital of Copenhagen by a mother, who came with her son, aged 1 year (Case 1). She was the mother of Wimmer's patient, who had died two years before, aged 9 years. His muscular disease had, if anything, become somewhat worse in the course of years. There were no other children than the boy now presented with the following history.

Case 1.—A. F. (fig. 1), son of a tailor, born October 18, 1914; examined November 23, 1915, at the Rigshospital. The parents were both well.

The mother, who had scarcely felt the movements of the child during the first pregnancy, felt them somewhat more during the second; the birth was normal, the patient was not asphyxiated.

Just after birth the movements were very slow and weak. The child could never lift his head or stand on his feet. This weakness had remained almost unchanged since birth; at any rate it had not got worse, the child had perhaps been somewhat more stiff. Mentally he was absolutely natural: he could say "da" (goddag—how do you do), but nothing else; he babbled, laughed and cried in a natural manner. He was suckled by his mother, and sometimes took a little milk soup and drank naturally. He never squinted, and could see and hear well. Stools normal, micturition free. The examination showed:—

Shape of the skull normal, only the parietal prominences strikingly large (this was also the case with the brother). The fontanelles admitted one finger-tip. The pupils reacted well to light. Very slight concomitant, convergent strabismus, the eye-movements free. Epicanthus on both sides. No facial

paralysis. The arms lay naturally, were moved freely but feebly. The patient could grasp and hold objects put into his hands, but when upright he could not lift his arms above the level of his shoulders. Considerable loss of tone, hyperflexibility about the shoulders, but not about elbows or, scarcely, about the wrists. Tendon reflexes were not obtainable. The patient reacted to pin pricks. The muscles of the arms were atrophic, but covered by a thick layer of subcutaneous fat.

The body.—Chest normal. Abdominal reflexes present on both sides. The spine straight, not rigid. When the patient sat straight up and somewhat bent forward, he was able to hold his head up, but as soon as he was pushed somewhat back, his head fell backwards, and he could not lift it himself (fig. 1). The patient reacted to pin pricks everywhere.

Lower extremities.—The muscles of thighs and legs seemed atrophic but covered with fat. Loss of tone, but no hyperflexibility. The patient could not stand on his feet, he fell on his kness and hips when he was propped up. The limbs were moved feebly but without striking slowness.

Knee-jerk and ankle-jerk could not be obtained. Plantar reflex normal. The patient reacted to pin-pricks on the legs. He moved his limbs without ataxia or tremor.

The examination did not otherwise show anything abnormal; no sign of rickets. The testicles had descended, there was no enlargement of the abdominal organs, and the stethoscopic examination was negative. Skiagrams of the extremities showed the muscles to be feebly developed, and the spaces between them seemed more pronounced than normal (intermuscular fat?).

No doubt this was a case of congenital familial progressive muscular dystrophy, and it is striking that the histories of the two brothers were similar. These cases closely resembled those described as amyotonia congenita. They were distinguished by (1) familial occurrence and (2) the fact that the disease does not improve, but that there is (in spite of the growth and development of the child) stagnation, relative retrogression.

While this case, like Wimmer's, was diagnosed as congenital familial muscular dystrophy, I propose mentioning some cases, first diagnosed as amyotonia congenita, later, in the light of additional facts, as heredo-familial disease, i.e., not amyotonia congenita.

Case 2.—A girl, born in August, 1914, died half a year old. The parents and three older sisters and brothers were well. The mother felt fœtal movements at the usual time and during the rest of the pregnancy. The child was born at term, labour was normal, and she was not asphyxiated. She was suckled by her mother for the whole of her life, and, apart from the disease of the nervous and muscular systems, she showed no symptoms till a few days

before death. Immediately after birth, the mother noticed that she did not move her legs at all and her arms only a little. She could only draw the back of her hand up to her mouth, but apart from this movement her hand

FIG. 2.--Case 2, C.

hung flaccid. Later, the mother noticed that the child could move her fingers and toes a little, and draw her legs up somewhat when she was raised. She could turn her head, but it dangled when she was made to sit up. She seemed to know her mother; when she got a little older, she laughed, babbled and

drank well. Her crying suggested that she had a difficulty in "weeping through." No convulsions.

On November 27 she was admitted to the children's department of the Rigshospital (University Hospital) of Copenhagen.

She (fig. 2) was well nourished. The head dolichocephalic. Tension of the fontanelles normal. Nothing abnormal about the eyes or fauces. Stethoscopic examination normal. The Bordet-Wassermann test was negative. Weight 5,480 grm. Temperature normal. Urine no abnormal contents. Hæmoglobin (Sahli) about 80 per cent. The stools were normal. X-ray examination of the upper and the lower extremities showed no abnormality of the bones, but the shadows of the muscles were seen distinctly, and were indicative of great atrophy as compared with the muscles of a normal child.

The patient seemed to have normal intelligence: she did not cry or tremble.

Neurological examination.—The pupils reacted to light. Ophthalmoscopic examination negative. Eye movements free, no ptosis, nystagmus nor strabismus. Right palpebral fissure somewhat more open than the left, especially on crying. Slight facial paresis on the right side. Chvostek's sign absent. The expression of her face, the smiling, crying and mimicry movements were natural. Facial and tongue muscles did not seem atrophic, but the temples were markedly sunken on both sides (atrophy of the temporal muscles?). The tongue was protruded incessantly and exhibited marked fibrillary movements.

Upper extremities.—Shoulder-joint abduction of 90 per cent.; outward rotation of humerus, flexion of the elbow-joint; marked pronation of the forearms; wrists slightly flexed; the four ulnar fingers slightly flexed; thumb hyperextended and very abducted. Movements of all the joints free, only supination and extension of the forearms somewhat limited. Hyperflexibility of the finger-joints, not of the other joints. Palpation of the arms gave the impression that the skin and subcutaneous tissues formed a very thick and easily displaced layer over atrophic muscles. The skin of the upper arm showed a very deep fold on the flexor aspect.

The patient could move her arms, apparently using all the muscles except the supinators of the forearms; all the movements were rather slow with little excursion; her hands seemed to be most active, but she was not inclined to grasp anything put in her hand. No tremor, no choreiform or athetotic movements. Pricks everywhere on the arms made her cry a little, but did not evoke defensive movements. Active vaso-motor reaction round the pricks. Tendon reflexes could not be obtained, nor the reflexes of Léri, although the patient showed reaction to pain. Tone much decreased. No cyanosis, œdema or other vaso-motor disturbances. No Trousseau tetany-phenomena.

The trunk.—When the child was placed erect, there was marked lordosis of the cervical spine and arcuate kyphosis of the back. No stiffness of the

neck. The patient did not try to lift her head, but turned it a little herself. The sterno-mastoid muscles seemed relatively powerful. The muscles of the back appeared to be somewhat atrophied. Respiration chiefly of the abdominal type, but also a little costal. Abdominal muscles seemed more feeble than normal. Abdominal reflexes could not be elicited. Reaction to pain on pricking. The chest somewhat narrow above, larger below. Sternum abnormally curved.

Lower extremities lay slightly flexed with normal abduction at the hip, slight flexion at the knee and very little supination at the ankle; position of the toes natural. Movements of the joints free, only abduction and flexion of the hips somewhat restricted. Tone diminished, no hyperflexibility (because of the atrophy?). Patellar, Achilles, and plantar reflexes could not be elicited. The condition of the skin and muscles resembled that of the arms. They gave the impression that bones with ill-developed muscles were loosely hung in a sack of skin and subcutaneous fat. The patient could move all her joints, but extension was slight and weak. No peroneal phenomenon.

The muscle reaction to the galvanic current was as follows: K.C.C.=5, A.C.C.=5·5, A.O.C.=10, K.O.C.$>$10. The responses were small and slower than normal. Faradic examination could not be undertaken.

The patient was discharged after two days. Afterwards she was treated as an out-patient with massage and electricity. Some insignificant movements in the extremities resulted, but in February the child developed an acute febrile disease and died in a few days. The parents consented to an autopsy by me at the Anatomical-Pathological Institute of the University. (I record my indebtedness to Professor Dr. Johs. Fibiger for his permission.)

The brain showed nothing macroscopically abnormal, but the spinal cord was strikingly thin and firm throughout. Sections were taken from different parts of the central nervous system, the sciatic nerve, the femurs and muscles for microscopic examination. The muscles were thin, pale and covered with a thick layer of subcutaneous fat.

The sections were hardened in 4 per cent. formaldehyde; some of the hardened sections were later treated with Weigert's brown chromic acid stain; others with chromic acid-acetic acid mixture and imbedded in celloidin and the paraffinoid of Claudius. The following methods of staining were carried out: Nissl's toluidin-blue staining, Weigert-Kulschitzky-Wolter's method for medullary sheaths, Bielschowsky's staining, Herxheimer's staining of fat. The muscle-sections were stained with v. Gieson-Hansen's and Herxheimer's methods.

Microscopic examination.—In the sections from the cerebrum and cerebellum, the great ganglia and pons, nothing abnormal was found. The pia mater of the spinal cord was normal; no wasting of medulla was seen in the appropriately-stained pictures and no augmentation of the glia; only in the anterior root exit zones was there a diminution of the medullary fibres, and corresponding to it a slight augmentation of the

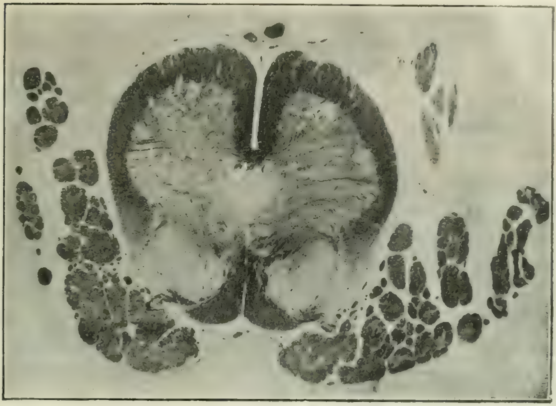

FIG. 3.—Spinal cord, stained with Weigert-Kulschitzky-Wolters method. Shows the degeneration of the anterior roots.

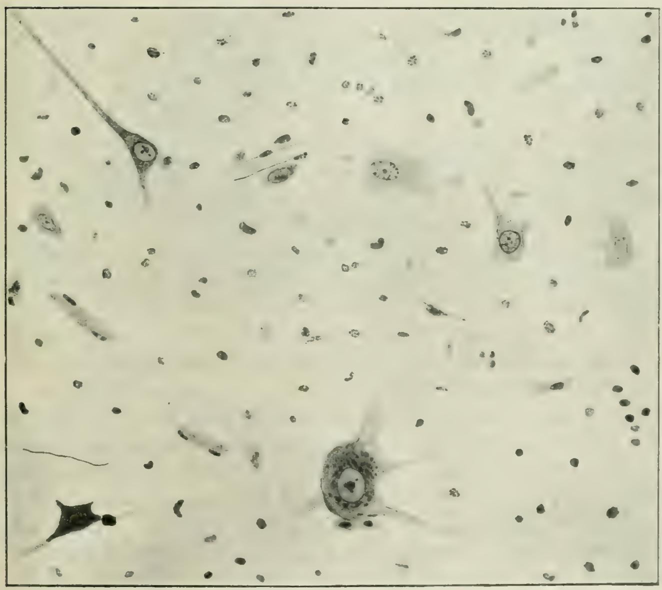

FIG. 4.—Anterior horn from the spinal cord of Case 2. Nissl staining.

glia. The anterior roots were very markedly degenerated; on the medullary-stained pictures they were white, while the posterior roots showed normal dark colour (fig. 3). The *nerve-cells*: In the anterior horns only a few of the great motor-cells showed normal size and form (fig. 4). Most of them were very shrunken, the protoplasm was less than normal, more angular and with a scanty number of prolongations. The protoplasm of these cells was ordinarily pale and homogeneous, without Nissl's bodies; in a few cases it was intensively stained, the cells showing a characteristic pyknoid condition. The protoplasm presented only a few vacuoles and there were no fat or pigment granules; the sections stained with acid fuchsin-vert-lumière showed a number of cells filled with acid fuchsin-stained granules. This observation must be judged with reserve; possibly these granules might be artificial. The nuclei of the cells were usually diminished, but less so than the cells as a whole; the nuclei were often eccentric. The chromatin of the nuclei was somewhat abnormal; it was distributed in comparatively large and intensely stained granules. Besides the nucleoli there were some small granules, stained in the same manner, perhaps expelled from the nucleoli. In some of the cells the nucleus appeared to be destroyed—a result, perhaps, of the preparation of the section. In a single nerve-cell there were two nuclei. In the most normal nerve-cells the Nissl bodies were preserved round the nucleus and destroyed in the peripheral part of the cell. The axis cylinders were relatively intensely stained and could be followed a long distance from the nerve-cells.

In each of the 10 μ sections there could be found only 1-2 relatively normal anterior horn cells; but in Clarke's columns the cells were normal, only with slight change of Nissl's bodies in the periphery; there were no shrunken forms.

The neuro-fibril sections showed well preserved neuro-fibrils in the best preserved nerve cells; in all the small and shrunken cells the neuro-fibrils were destroyed and appeared only as small granules.

While the amount of the glia in the white matter (seen apart from the anterior root zone) was absolutely normal, there was a considerable increase of glia in the grey matter. There was a greater number of glia-cells than normal in both the anterior horns, and there was a great amount of glia-fibres passing through the anterior horns in all directions, most dense in the anterior part, but otherwise evenly distributed in the anterior horns. There was nowhere any dense accumulation of glia. The glia-cells were nearly all of the fibrillary type; no ameboid,

vacuolized or giant forms were seen. There was no increase of the satellite cells round the nerve-cells; medullary fibres and glia were evenly distributed throughout the spinal cord.

In the fat-stained sections no fat or lipoid granules were seen in the glia-cells or nerve-cells.

The vessels were everywhere normal; there was nowhere any

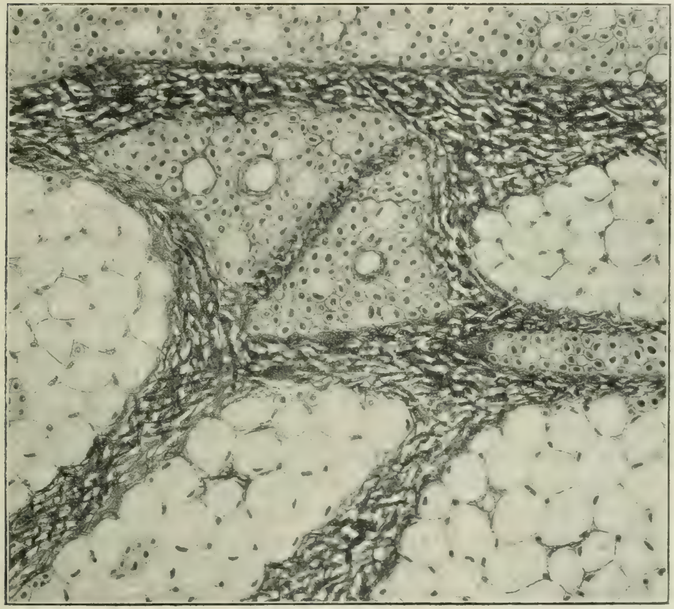

FIG. 5.—Muscle from Case 2, stained with v. Gieson-Hansen's method, showing normal and atrophic muscle fibres and connective tissues.

perivascular infiltration with lymphocytes, leucocytes or plasma-cells. There seemed, however, in the anterior horns, to be an increase in the number of small vessels.

The above-mentioned section of the sciatic nerve showed a moderate diffuse increase of the connective tissue but no sign of inflammation.

The appearance of the muscle examined was characteristic (fig. 5). Some of its fibres were perfectly normal; but a considerable number of muscle fibres were very thin, without obvious striation and with a

single central nucleus in every fibre. There were no hypertrophic fibres, but here and there transitory forms between atrophic and normal. In these transitory forms the nuclei were somewhat eccentric. The protoplasm in the atrophic fibres usually stained more darkly than that of the normal ones, and no transverse or longitudinal markings were seen.

The normal and atrophic fibres were separately distributed ; here and there, however, small groups of atrophic cells were seen in the normal bundles and normal cells in the atrophic bundles. Between the bundles there was much connective tissue. But there was no increase of the perimysium between the single fibres, no increase of the vessels, no sign of inflammation, no leucocytes. The atrophic fibre-bundles contained many more nuclei than the normal bundles. This increase of nuclei was, however, only relative, a consequence of the atrophy of the protoplasm. The transverse sections of the muscles showed the atrophic muscle-fibres to be far more numerous than the normal.

The clinical and post-mortem findings established the diagnosis : amyotonia congenita. It was a case of congenital muscular weakness with hypotonia of the muscles and loss of the tendon reflexes. The fact that there was atrophy did not tell against the diagnosis, for in many of the recorded cases of amyotonia congenita there had been atrophies. The post-mortem findings absolutely coincided with the cases of Rothmann, Archangelsky-Abrikosoff and Marburg, only the absence of hypertrophic fibres distinguished it from the cases of Badouin, Reyer-Helmholtz, Collier-Holmes, Griffith-Spiller, Laignel-Lavastine-Voisin and Kaumheimer. There was no history of heredity ; the three elder sisters and brothers of the patient were well.

Now, however, five years later, it appears that the disease was nevertheless familial ; in August, 1919, the mother applied to the polyclinic for children at the Rigshospital concerning a younger brother, whose history is as follows :—

Case 3.—M. J. C., born March 25, 1919, son of a coffee merchant. The three elder sisters and brothers were well. Apart from the dead sister there was no history of nervous disease in the family.

The mother had suffered from slight influenza during the first month of pregnancy, which, however, was normal. She felt fœtal movements at the normal time and during the rest of her pregnancy.

The patient was born normally at term ; his weight at birth was 3,500 grm. During the first five months he was suckled, but after that time he was given milk, milk-soup and gruel. His growth was normal.

The mother noticed soon after his birth that the child moved his legs very little and was absolutely flaccid. This flaccidity did not disappear; when he was about half a year old he could not lift his head from the pillow, but only turn it a little. He moved his arms freely, but not as actively as a normal child; he could grasp different things, but hold them with difficulty. He could lift his legs under himself and move his head from his pillow. There was no history of convulsions or fever. His mental development corresponded with his age; he was interested in his surroundings, smiled and was quiet and amiable.

At the hospital the patient was seen to be well developed and nourished, not anæmic. No glandular swelling of the neck, no swelling of the thyroid gland. Weight 6,750 grm., length 64 cm. The fontanelles 2 by 2 cm.; teeth 0.0. Urine normal. V. Pirquet and Bordet-Wassermann reactions negative. Genital organs normal. The child had blue eyes, light hair, and was dolichocephalic.

Ophthalmoscopic picture normal. Pupils reacted well to light. Eye movements free. No strabismus, ptosis or nystagmus. The child followed moving objects with his eyes and turned his head in response to noise. No facial paralysis. No Chvostek's sign. He smiled sometimes; his expression was natural. Considerable fibrillary movements of his tongue. No paresis of the soft palate.

He could move his head from side to side but never tried to lift it from the pillow. When he sat semi-erect, there was lordosis of the cervical spine, so that his head sank between his shoulders. Though it tended to fall forwards or backwards, the child tried to keep it straight. The dorsal and lumbar spine showed a slight arcuate kyphosis. Respiration was mainly abdominal, only a little thoracic. The abdominal muscles were weak, and the child was unable to contract them. Abdominal and cremasteric reflexes could not be elicited. Only a little pain-reaction on pricking the trunk.

Upper extremities.—Position normal. No atrophy was seen, but on palpation through the thick layer of fat the muscles seemed atrophic. The child moved his limbs freely at all the joints, but the movements were weak. Tone seemed diminished, but there was no hyperflexibility at the joints. Tendon reflexes could not be elicited. He reacted to pricks with strikingly small movements and a little grunt, but no crying.

Lower extremities lay rotated outwards, a little flexed at the knees. There was *pes planus* on both sides. The foot formed a rectangle at the ankle with the heels prominent. The child was able feebly to flex and extend his lower limbs at the ankles and toe-joints. Very feeble flexion and extension at the knee, slight movement at the hip. When the leg was lifted, it fell immediately flaccid. When the child was propped upright, his knees gave way and he made no attempt to straighten them. Tone of the leg muscles was scarcely diminished, but there was hypotonia of the hip- and thigh-muscles. No hyperflexibility, but it was rather difficult to move the extremities outwards. Patellar and Achilles reflexes could not be elicited. Plantar reflexes of normal type but feeble. The child reacted by crying when the lower extremities were pricked.

Electrical reactions: K.O.C. = 8; A.O.C. = 5; A.C.C. = 2·5; K.C.C. = 4.

No doubt this boy suffered from the same disease as his dead sister. The pseudo-heredity of congenital syphilis could certainly be excluded. We must, therefore, diagnose these two as cases of familial congenital muscular dystrophy, which in no way differ from Wimmer's and my two cases of congenital muscular dystrophy. Only the progress of the palsies and the atrophies was more marked in Wimmer's case than in the two cases described here.

I have examined some more cases, diagnosed as amyotonia congenita, in the children's department of the Rigshospital. Further examinations of the family histories showed that the disease was familial.

Case 4.—N. H. B., son of a farmer, born October 7, 1915, treated in the children's department of the Rigshospital, January 19 to February 6, 1916.

No case of muscular or nervous disease in the family (cf. *infra*). The parents were well, an elder brother was sane (for description of the family, see figs. 6-7). The father's brother had disseminated sclerosis (the diagnosis certified at the Rigshospital's clinic for nervous diseases).

The child was born naturally at term. The mother had felt fœtal movements during the last half of pregnancy. The child was suckled by its mother during the first three months, afterwards he was given boiled milk.

The mother had noticed that the child since birth always held its hands in a certain position, flexed at the wrists, extended at the metacarpo-phalangeal joints and flexed at the interphalangeal joints. He could move his arms well and lift them, and he could move his fingers, but not straighten them out. There was also a somewhat abnormal position of the left foot. Further, during respiration, there was always marked retraction of the thorax round the ensiform process. The mental condition was normal.

On admission to the Rigshospital the child was small and flaccid, somewhat apathetic during the examination, and only on inspection of his throat did he cry a little. He was fairly well nourished. Weight 4,750 grm. The fontanelles 3 by 3 cm., somewhat sunken. No teeth. Stethoscopic examination normal. v. Pirquet's reaction negative. The pupils reacted to light. No atrophy of the facial muscles. When crying there was some tendency to draw the mouth to the left; there was, however, no marked facial paralysis. No Chvostek's sign. Fauces normal.

He could not lift his head from the pillow nor move it from side to side. When he was placed erect his head fell flaccidly backwards. The neck muscles seemed atrophic. Respiration chiefly abdominal; during respiration the lower part of the sternum was drawn deeply in and from it a deep sulcus was continued to both sides of the costal arch. Abdomen somewhat prominent, especially the epigastrium.

Upper extremities.—Right upper arm was abducted (about 90°), the forearm flexed to 90° and permanently pronated. Hyperextension at the metacarpo-phalangeal joints. The fingers were flexed. Left upper arm also somewhat

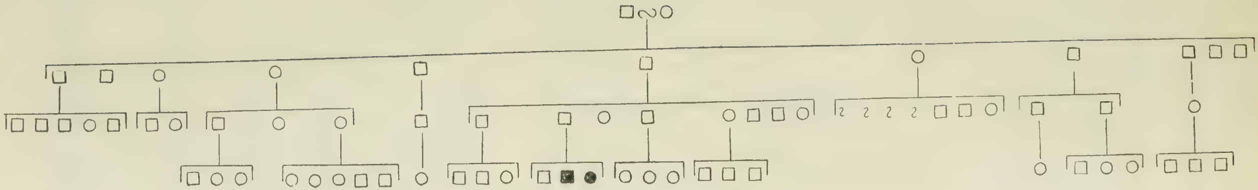

FIG. 6.—Case 4, father's family.

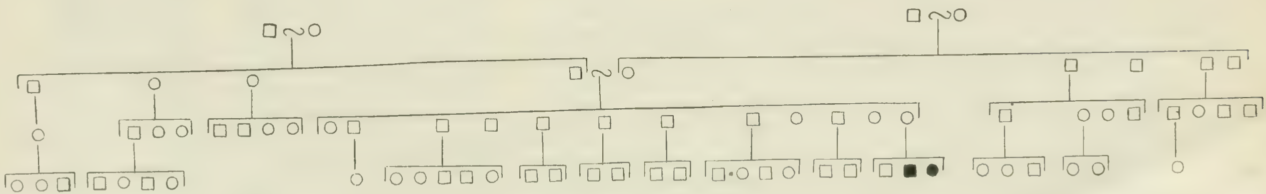

FIG. 7.—Case 4, mother's family.

abducted, but less than the right; the forearm flexed and pronated. Hand and fingers as on the right side. Passive movements could be executed normally. The patient held the left elbow a trifle flexed, rotating the right forearm and moving the right shoulder only a little. The muscles did not seem to be strikingly atrophic, but it was difficult to judge because of the abundant subcutaneous fat. No radial, nor Trousseau's phenomenon.

Lower extremities lay a little abducted and rotated outwards. Left foot lay in a slight valgus position; right foot had an arched instep and was rather in an equinus position. After trying to rotate the thigh inwards, the limb fell back to its original position. Passive extension at the hip was normal, there was no hyperflexibility. The knee could only be moved about an angle of 90°; the ankle could be moved freely. The patient could not himself lift his limbs from the bed or bend his knees, and he could only flex his ankles a little. The trophic condition of the muscles was difficult to judge because of the abundant fat. Sense of pain scarcely diminished. Patellar and Achilles reflexes could not be elicited. Plantar reflexes very feeble, of normal type.

Electrical examination of the left median nerve showed: K.C.C. = 6·5; A.C.C. = 4·5; A.O.C. = 10; K.O.C. = 10.

The contractions were very slow and feeble. The left peroneal nerve: K.C.C. = 6; A.C.C. = ? (10): A.O.C. = 13; K.O.C. = 13.

X-ray examination in the upper and lower extremities showed nothing abnormal in the bones. The muscles, especially the crural muscles, seemed somewhat feebly developed: the atrophy was, however, not very marked.

After the patient had been 14 days in hospital signs of pneumonia on the left side suddenly appeared, and death occurred after two days. An autopsy was not permitted.

The diagnosis was: Amyotonia congenita. As I had ascertained that Case 2, which was also diagnosed as amyotonia congenita, was really one of hereditary disease, I searched for further data about the family of this case from an uncle who was a doctor. He gave me the surprising information that the mother of the child two years after the death of the patient had a daughter, who a short time after birth had showed a paralysis just like that in the patient here described; the child died half-a-year old of pneumonia. After that there is no doubt that the disease, tentatively diagnosed as amyotonia congenita, was really the same congenital familial muscular dystrophy as in Cases 1, 2 and 3.

The following case showed the same clinical and pathological picture as that already described. The familial occurrence is here somewhat more doubtful.

Case 5.—G. S., daughter of a farmer, born April 6, 1918, admitted to the children's department of the Rigshospital, July 19, 1918; died there August 6,

1918. A sister of the father's father had from birth been paralysed in arms and legs, was wheeled about in a Bath chair, died about 8 years old. Otherwise no cases of paralysis in the family. The patient had no sisters or brothers.

The child was born in breech-presentation by extraction after version. She was given both mother's milk and tapioca-soup and milk. Even from birth the muscles of the neck and the extremities were flaccid, she could move her arms and legs very little and not extend them at all. She moved her fingers and toes quite well. The left arm was moved somewhat better than the right. The head could be moved only very slightly from side to side. The movements of the lower extremities improved, and by and by the patient could draw them somewhat up and extend them again. Never cramps or convulsions. The child was always active and happy and drank well.

The examination on admission showed the child to be mentally well developed. Expression of the face normal. Cried vigorously. She was well covered with fat. Stethoscopic examination negative. The fontanelles 4 by 3 cm., tension normal. The cranium firm.

The temporal muscles seemed slightly atrophic. Fibrillary contraction of the tongue. Cervical muscles atrophic. When she lay down she could move her head only a little from side to side. When she sat, her head fell forwards or backwards. Thorax paralytic, the respiration only of abdominal type. Abdomen soft.

Upper extremities.—Shoulder and pectoral muscles atrophic, the other muscles seemed also atrophic, but less so; there was some difficulty in palpating through the subcutaneous fat. The muscles were flaccid. Considerable hyperflexibility at the shoulder-joints, not at the other joints. Forearms pronated. The patient moved the left arm better than the right. This could only be moved about ten degrees at the elbow and hardly at all at the shoulder. Left arm could be lifted a little from the bed. The power of the hands was somewhat better, the patient could hold a finger feebly. Reflexes could not be elicited.

Lower extremities.—The muscles were flaccid and seemed atrophic, but their condition was somewhat difficult to judge because of the abundant fat. The child moved her legs a little better than her arms, best at the ankles and toes, but very feebly. Reflexes could not be elicited. The sense of pain was everywhere normal.

X-ray examination of the extremities showed the muscles of all the extremities, chiefly those of the arms, to be somewhat atrophic. Bordet-Wassermann reaction negative. v. Pirquet reaction very slightly positive. The urine contained no sugar.

In hospital the child drank well at first, cried somewhat, but was otherwise rather quiet. After ten days pneumonia developed on the right side. Her respiration became short and shallow, and she coughed without expectorating. In spite of treatment with steam, camphor, and digalen injections, the patient collapsed and died August 6, 1918. At the autopsy broncho-pneumonia and purulent bronchitis of both lungs were found.

I examined the spinal cord microscopically, but not the muscles. The microscopic examination showed changes corresponding closely with those of my former case. In the different segments of the spinal cord a few of the anterior horn cells were normal, with a beautiful tigroid staining. But most of the anterior horn cells were atrophic, angular, and showed rubbed-out tigroid staining, the protoplasm being homogeneous or very finely granulated. Nuclei and nucleoli were, however, normal. The cells of Clarke's column were relatively more normal, as they were not diminished in size; they showed only in the periphery normal Nissl-granules; all the part round the nucleus was homogeneous. In the medullary-stained preparations the anterior roots were conspicuously degenerated, but the posterior roots retained their normal colour. In other respects they showed no degeneration of the white substance.

Pathologically this case resembles the first described, clinically also the resemblance is great. The familial history is somewhat more dubious, in that the paternal aunt mentioned might have been paralysed from a poliomyelitis or other disease.

In the following case the familial condition was not found in a brother or a sister, but in other, more distant relatives. The diagnosis, therefore, was dubious: either it was amyotonia congenita or congenital progressive muscular atrophy. Clinically the case was like the others.

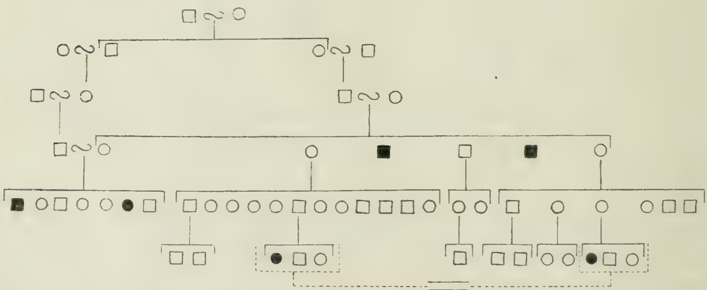

FIG. 8.—Case 6, father's and mother's family.

Case 6.—E. B. C., daughter of a lawyer, born April 12, 1917, treated in the children's department of the Rigshospital, July 10 to July 21, 1917.

The mother's mother gave me the following information: The parents

were cousins. The two brothers of the mother's mother (and the father's mother) died about 8 months old; they were said to have been paralysed from birth, one of them in the upper, the other in the lower limbs. Further information could not be obtained, as they died fifteen years before the birth of the mother's mother. The father's mother's and the mother's mother's elder sister was related to her husband (fig. 8). She had had a son and a daughter, both of whom had been paralysed shortly after birth and had died suddenly about 8 months old in connection with catarrhal infections.

The mother was well during pregnancy, and had certainly not shown any sign of poliomyelitis. She felt fœtal life during the last part of pregnancy. Labour was normal, lasting only three hours. The weight of the child at birth was 3,500 grammes.

When the child was one month and a half old, the grandmother remarked that her legs were very flaccid. About fourteen days later the grandmother remarked that her arms were singularly abducted and flexed at the elbows, and that the flaccidness of the legs was not marked. The condition seemed, however, again to improve a little. She was suckled by her mother during the first month and a half, after that milk and barley water were given.

On admission to hospital the child was seen to be small and very flaccid, but well nourished. Her head was dolichocephalic, her fontanelles normal. Her intelligence seemed normal.

Pupils reacted well to light. Ophthalmoscopic examination negative. Eye movements free, now and then a little strabismus, no ptosis or nystagmus. Temporal muscles somewhat flattened. No facial paralysis. No Chvostek's sign. Facial expression smiling, and mimicry natural. The tongue did not deviate on protrusion, but it showed some fibrillary movements. Sucking energetic.

Upper extremities usually abducted to 90°, flexed at the elbows and rotated outwards. Wrists were slightly flexed. Free passive movements at all the joints, hyperflexibility in them all, especially the shoulders. The muscles, on palpation through the abundant fat, seemed very atrophic. The patient moved the elbows, both spontaneously and when pricked. The range of the feeble movements at the shoulders was limited. No tendency to grasp things. No tremor, athetosis or choreiform movements. Sensibility seemed everywhere diminished, the child did not cry even when pricked deeply, but did so when the wrist was flexed roughly. No vaso-motor reaction about the pin pricks. Tendon reflexes could not be elicited.

Trunk.—When the patient lay down, she only moved her head from side to side, but did not try to lift it. When she sat up somewhat, she could not hold her head up, it fell forwards or backwards. In spite of this she tried to lift it up. Muscles of the back were very flaccid; when she was set up there was a somewhat arcuate kyphosis of the dorsal and lumbar spine and lordosis of the cervical spine, so that her head sank down between her shoulders. Her respiration was chiefly abdominal. Abdominal reflexes could not be elicited.

No pain reaction to pricks on the body. No vaso-motor reaction. There was incontinence of urine.

Lower extremities.—Position practically normal. Tone everywhere diminished, and there seemed to be some hyperflexibility. Tendon reflexes absent. By pricking the sole of the foot the patient reacted both by contraction of the quadriceps and by defensive movements of the foot. She could actively but feebly move most of her joints.

Lumbar puncture yielded blood-stained fluid. Both this and the blood showed a negative Bordet-Wassermann reaction.

Electrical reactions: K.C.C. = 4, A.C.C. = 5·5, A.O.C. > 10, K.O.C. > 10.

In hospital the child appeared mentally normal, smiled and babbled.

After ten days she was sent home, where she died after a short time. No autopsy.

A résumé of the cases and of Wimmer's case shows the following points :—

Familial incidence occurred in six cases undoubtedly, and possibly in one case. In two cases, Wimmer's patient and his brother, the mother had not felt fœtal life or only very slightly; in three other cases the mother felt life in a normal manner in due time. In one case the child was born in breech presentation by extraction after version; in all other cases birth was normal. In five cases the disease was noticed immediately after birth, in the sixth it was noticed about fourteen days later, and in the seventh one month and a half after birth. It is, however, very probable that the disease in these two cases also was congenital, but was not detected at once.

The pareses were extensive in all the patients. In one of the patients there was a trace of facial paresis; the cranial muscles, especially the eye muscles, seemed otherwise to have been spared. As a rule, the upper and lower extremities were affected in similar degree and more centrally than peripherally. The trunk muscles were also usually affected, all the children collapsed when propped up, the head fell forwards or backwards, and none of the children could lift the head from the pillow. The respiration was mainly abdominal, in several of the children the thorax seems to have been completely paralysed.

Tone was diminished as in amyotonia congenita, but the hyperflexibility of the joints, which is common in amyotonia, was observed only in the fingers and wrists and, in two cases, in the shoulder joint. The trophic condition of the muscles was often difficult to judge because of the abundant fat. Both palpation and X-ray examination showed great

and extensive muscular atrophies. Fibrillary contractions of the tongue were observed in three patients only. Tendon reflexes were absent in all. The sensory phenomena were variable, as a rule the sense of pain was intact; in two of the patients, however, it showed a diffuse limitation. None of the patients showed any sign of syphilis.

The pathological findings in two cases showed marked conformity with most of the recorded cases of amyotonia congenita: a degenerative condition of the nerve-cells, anterior roots and muscle-fibres without any sign of inflammation and without any lesion of other parts of the spinal cord. Marburg suggests that this condition is due to intra-uterine poliomyelitis. There does not seem to be sufficient reason for accepting this very interesting theory. Marburg supposes that amyotonia is the result of a pre-existing, not an active, process in the spinal cord. It seems, however, impossible to decide if an atrophic pyknoid condition of the nerve-cells is a result of a pre-existing inflammatory process or of a very slow degeneration as in the muscular dystrophies. Marburg is right in assuming that these findings cannot be traced to arrested development. But he cannot point out any certain differences from the progressive muscular dystrophy of the Werdnig-Hoffmann type. To demonstrate the conformity of the recorded cases, both clinically and pathologically, with the progressive muscular dystrophy of the Werdnig-Hoffmann's type, I report the following case.

Case 7.—P. L., born January 27, 1917, son of a job-master, treated at the Children's Department of the Rigshospital, May 30 to June 28, 1918, and March 16 to September 19, 1919. A sister of the patient, aged 5 years, completely well. A father's brother suffered from a congenital paralysis and died at the age of 5 years. Two cousins of the father (his father's sister's sons) had a similar disease; one of them died 16 years old of cerebral affection, the other died 11 months old.

The patient was born naturally, labour lasted only one hour. During pregnancy the mother felt normal fœtal movements at the usual time. He was suckled by his mother for three months, developed normally, smiled and moved himself about freely during the first eight months. At eight months it was remarked that he could not remain erect when raised, later there was no change in this respect. He seemed also from that time to move his legs less well than before; he improved somewhat after treatment with massage. Otherwise he was perfectly well, there were no convulsions or tremors, no squint. The examination (in my private practice) February 6, 1918, when the child was 1 year old, showed the patient to be very fat. Upper extremities, which were hypotonic but not hyperflexible, were moved freely. Lower extremities very

flaccid. Tendon reflexes absent. When pricked with a needle he moved his legs slightly. The crural muscles reacted both to the faradic and the galvanic current somewhat slowly, especially on kathode-make.

On admission to the Children's Department of the Rigshospital, May 30, 1918, the child was well nourished. Left-sided cryptorchism; examination of the organs showed nothing abnormal. Urine normal. v. Pirquet reaction negative. Hæmoglobin (Sahli) 87 per cent. Weight 8,620 grm. Fontanelles closed; 4/4 teeth. Stethoscopic examination of the heart and the lungs negative. The child was quick, at one examination only somewhat dull. Nothing abnormal in the cranial nerves.

Trunk.—When he was placed erect, the muscles of the neck and back were flaccid. The thorax was soft and retracted during respiration. Abdominal reflexes present.

Upper extremities were moved freely. The muscles were somewhat flaccid and atonic. *Lower extremities*: The muscles everywhere flaccid and atrophic. He could only draw his legs up a little. Movements of the feet and toes were apparently absolutely free. Tendon reflexes could not be elicited. Plantar reflexes normal.

Electrical reactions: K.C.C. = 5, A.C.C. = 6.15, A.O.C. > 10, K.O.C. > 10.

Later examinations, March 18 and June 22, showed that he could not draw his legs up as at first. He seemed during his stay in hospital to be somewhat backward mentally.

March 16, 1919: The child was again admitted for pneumonia. The mother said that he had recently been rather feeble in spite of treatment with massage. Two days before admission he became febrile and dyspnœic. On admission there were signs of pneumonia on the right side, and the child died in twenty-four hours.

The autopsy showed broncho-pneumonia of both the lungs and purulent bronchitis.

Microscopic examination of spinal cord.—In the lumbar cord the cells of the anterior horns were very markedly diminished in number. A few were quite normal, and a few were very atrophic, pale, thin and angular. In the dorsal and cervical regions the appearance was somewhat different: there were hardly any normal cells to be seen, but a considerable number of abnormal nerve cells. All these cells were diminished in size, angular and slender, but not pale; on the contrary, they were dark-coloured, with pyknoid protoplasm which hid the nucleus. In the dorsal region the cells of Clarke's column were normal in size and shape, but without staining of Nissl's granules.

It should be noted that the difference between my six cases and the progressive muscular dystrophy of Werdnig-Hoffmann's type is very little, both clinically and pathologically. But it must be emphasized

that my cases are congenital and so far represent a great rarity, for only very few cases of congenital muscular dystrophy seem to have been recorded. This disease would seem to be much more rare than amyotonia congenita, of which more than sixty cases have been published.

We will, however, make the following comparisons :—

(1) Amyotonia congenita, according to the original descriptions, seems to be a rather benign and not an hereditary disease, and does not seem to leave any defects in adults.

(2) In eight of the eleven cases which have come to autopsy, there have been severe atrophies of the anterior horn cells and muscles.

(3) In two of my cases of hereditary congenital muscular atrophy corresponding changes were seen.

(4) These changes resemble, on the whole, the changes in Werdnig-Hoffmann's progressive muscular atrophy.

(5) In my six cases the disease clinically resembled closely amyotonia congenita, and only later information showed that it was a familial disease.

I would emphasize the radical pathological difference existing between the heredo-familial diseases and other diseases and, remembering that every heredo-familial disease in a certain number of cases is found isolated, I would suggest the following explanation as the most probable :—

The cases which have been described as amyotonia congenita represented really two different diseases :—

(1) One of these, amyotonia congenita (myatonia congenita, Oppenheim), is a benign disease, which consists of a congenital hypotonia, hyperflexibility and weakness, but *no atrophies*. If the patient does not die from intercurrent diseases, it may be assumed that he is cured. It is not familial and can possibly be considered as a retarded development of the muscles.

(2) From this true amyotonia congenita must be separated the following : The cases first described by Beevor[1], Sorgente, Silvestri and Skoog. Secondly the cases in which, at the autopsy, atrophy of the anterior horn cells and muscles was found. They are Rothmann's, Reyer-Helmholtz's, Archangelsky-Abrikosoff's, Marburg's, Collier-Holmes', Griffith-Spiller's, Laignel-Lavastine-Voisin's and Kaumheimer's.

[1] Originally considered as myopathy, later as amyotonia.

All these, with Batten's cases, Howard's case, Wimmer's case and Jendrassik's case, and the six cases which I have described, must probably be considered as cases of congenital familial progressive spinal muscular atrophy, a type which is most related to Werdnig-Hoffmann's type, but is congenital. This disease has a certain clinical resemblance to amyotonia congenita, but differs from it in that the muscles show a marked atrophy, demonstrable by the X-rays, and in that it has a tendency to progression or at any rate not to improvement, and in that, for a certain number of cases, it can be shown that the disease is familial.

My best thanks are due to Professor C. E. Bloch for permission to examine cases in his wards and for the interest he has shown in my labours. I also wish to thank Professor Dr. Viggo Christiansen for permission to publish the first case from the neurological polyclinic of the Rigshospital.

REFERENCES

ARCHANGELSKY und ABRIKOSOFF. "Ein Fall von Myatonia congenita Oppenheim mit Autopsie," *Archiv für Kinderheilk.*, 1911, Bd. lvi, S. 101.

BATTEN. "Three Cases of Myopathy, Infantile Type," *Brain*, 1903, vol. xxvi, p. 147.

Idem. "Progressive Spinal Muscular Atrophy of Infants and Young Children," *Brain*. 1910-11, vol. xxxiii, p. 433.

Idem. "Myopathy, Infantile Form of Landouzy-Dejerine's Type," *Proc. Roy. Soc. Med.*, 1914-15, vol. viii, Neurol. Sect., p. 70.

BATTEN and GORDON HOLMES. "Progressive Spinal Muscular Atrophy of Infants" Werdnig-Hoffmann's type, *Brain*, 1912-13, vol. xxxv, p. 38.

BEEVOR. "A Case of Congenital Spinal Muscular Atrophy, &c.," *Brain*, 1902, vol. xxv, p. 85.

CHÉNÉ. "L'atonie musculaire congénitale (maladie d'Oppenheim)," *Thèse de Paris*, 1910, contains 57 references.

COLLIER. "Case of Amyotonia Congenita," *Proc. Roy. Soc. Med.*, 1914-15, vol. viii, Neurol. Sect., p. 71.

COLLIER and HOLMES. "The Pathological Examination of Two Cases of Amyotonia Congenita, with the clinical description of a fresh case," *Brain*, 1909, vol. xxxii, p. 187.

COLLIER and WILSON. "Amyotonia Congenita," *Brain*, 1908, vol. xxxi, p. 1.

CONCETTI. "Ancora sulla atonia muscolare congenita di Oppenheim, in specie dal punto di vista anatomo-pathologico," *Riv. di clin. pediatr.*, 1913, t. xi, p. 1.

FEARNSIDES, E. G. "Case of Amyotonia Congenita (? Atonic Form of Cerebral Diplegia)," *Proc. Roy. Soc. Med*, 1914-15, vol. viii, Neurol. Sect., p. 49.

FOOT. *Amer. Journ. Diseases of Children*, 1913, vol. v, p. 359.

GRIFFITH and SPILLER. "Amyotonia Congenita: a Clinical and Pathological View," *Amer. Journ. of Med. Sciences*, 1911, vol. cxlii, p. 165.

GUTHRIE. *Trans. Clinical Society*, 1899, vol. xxxii, pp. 262, 263.

JENDRASSIK. *Deut. Zeitschr. für Nervenheilk.*, 1902, Bd. xxii, S. 444.

Idem. In Lewandowsky's "Handbuch der Neurologie," Bd. ii.

KAUMHEIMER. "Zur Pathologie und Klinik der Myatonia congenita" (Oppenheim), *Jahrbuch für Kinderheilk.*, 1913, Bd. lxxviii, Suppl., S. 170.

LAIGNEL-LAVASTINE et VOISIN. "Note histologique sur la myatonie congénitale," *Revue neurologique*, 1913, t. xxvi, p. 46.

MARBURG. "Zur Klinik und Pathologie der Myatonia congenita" (Oppenheim), *Arbeiten aus dem Neurol. Inst. Wien.*, 1912, Bd. xix, S. 133.

REUSS, A. "Die Krankheiten der Neugeborenen," Berlin, 1914.

SKOOG. "Myatonia congenita," *Zeitschr. f. d. ges. Neurol. u. Psychiat.*, 1914, Original, Bd. xxvii, S. 357.

SPILLER. "The Relation of the Myopathies," *Brain*, 1913-14, vol. xxxvi, p. 75.

THORSPECKEN, "Beitr. zur Kenntnis der Myatonia congenita" (Oppenheim), *Jahrbuch für Kinderheilk.*, 1912, Bd. lxxvi, S. 300.

WHAIT. "A Case of Amyotonia Congenita," *Proc. Roy. Soc. Med.*, 1907-8, vol. i, Neurol. Sect., p. 61.

WIMMER. "To Tilfælde af kongenit Muskellidelse hos Born," *Nordisk Tidsskrift for Terapi*, 1905-6, Bd. iv, S. 193.

Idem. "Zwei Fälle von congenitalem Muskelleiden bei Kindern," *Archiv für Psychiatrie*, 1907, Bd. xlii, S. 960.

ZIEHEN. "Beziehungen zwischen angeborenen Muskeldefekten, infantilem Kernschwund und Dystrophia musculoris progressiva infantilis." *Berl. klin. Wochenschr.*, 1908, Bd. xlv, S. 1557.

Publications Recently Received.

[*Notes on a Book under this heading do not preclude a subsequent review.*]

The Conduction of the Nervous Impulse. By KEITH LUCAS, SC.D., F.R.S. Revised by E. D. ADRIAN, M.D., M.R.C.P. Pp. 102. London: Longmans, 1917.

This remarkable work made its appearance during one of the most difficult periods of the war, and failed to attract the notice it deserved. Dr. Keith Lucas delivered a course of seven lectures at University College, London, in the spring of 1914, and intended to publish them as a monograph. At the outbreak of War he joined the Army and was posted to the Royal Aircraft Factory. There he was killed in an aeroplane accident on October 5, 1916. After his lamentable death, Dr. Adrian completed the manuscript, and wrote several of the chapters either from notes left by Dr. Lucas, or from his knowledge of the views held by his fellow-worker and friend. Most practical neurologists are not acquainted with the revolution created by these researches in our knowledge of the nature and conduction of the nerve impulse. This little book is therefore greatly to be welcomed, because the views of Keith Lucas are here put together in an accessible form, and expressed in the simplest possible language. It is worthy of close study by all who are interested in the modern views of the physiology of the peripheral nervous system.

Travaux neurologiques de Guerre. By GEORGES GUILLAIN and J. A. BARRÉ. Preface by Professor PIERRE MARIE. Pp. 463. Paris: Masson, 1920.

In this volume the authors have collected together their valuable contributions to the literature on the effects of injuries of the nervous system in war. They cover a vast field, for sections are devoted to wounds of the head, of the spinal cord and of peripheral nerves, the study of reflexes in the lower extremities by graphic methods, and a variety of other subjects. Much of their work was carried out close to the firing line and is of especial interest, since it deals with the early effects of acute lesions of the nervous system. For example, they were able to study the results of transection of the spinal cord in fifteen patients almost immediately after they were wounded, and their description of the findings is unique. The chapter on Concussion of the Nervous System is particularly fascinating. No less than 789 patients with injuries of this class passed through their hands, and many rare conditions, such as epileptic crises, generalized contracture and disorders of co-ordination

resembling those of disseminated sclerosis, were observed during the early stages of the illness.

The book is a record of investigations carried out with extreme care and under great difficulties by skilled observers and fully merits the laudatory remarks of Professor Pierre Marie in his preface.

Mammalian Physiology, a Course of Practical Exercises. By C. S. SHERRINGTON, M.D., D.Sc., F.R.S. Pp. 156, with many illustrations. Oxford: Clarendon Press, 1919.

This remarkable book purposes to set out a series of practical exercises for a class in physiology; but the novel feature is the extensive use made of the decapitate or decerebrate mammalian preparation. This takes the place of the frog in most of the experiments, and the student is able to make his observations on a warm-blooded animal, which has been pithed at a higher or lower anatomical level. "The actual performance by the student," says the author, "of some few such main experiments gives him, I am convinced, a better insight into their general significance and into the problems they touch, than does any mere inspection at the demonstration, however skilfully conducted." All the graphic records are those made by students themselves, and in many cases their names are immortalized at the foot of the curve. Many of us can only look with envy on the happy members of such a class.

Mikroskopischer Atlas des menschlichen Gehirns. Herausgegeben von Professor Dr. G. FUSE und Professor Dr. P. C. von MONAKOW. I. Die Medulla Oblongata. Consisting of plates, 44 × 59 cm. Zurich: Orell Füssli, 1916.

This atlas contains six large plates representing transverse sections of the medulla oblongata. They were drawn by Professor G. Fuse, of Japan, in the Institute of Professor von Monakow. On each plate the parts are named so that an explanatory text is unnecessary. The various structures have as far as possible been checked by secondary degenerations and comparison with other collections of similar microscopical preparations. If the subsequent portions of this work approach in accuracy and fulness the present atlas, it will be a most valuable guide to future workers in organic pathology of the nervous system.

Les Médications psychologiques. I. L'Action morale, l'Utilisation de l'Automatisme. Pp. 343. II. Les Économies psychologiques. Pp. 307. III. Les Acquisitions psychologiques. Pp. 494. Three volumes paged independently with a general index at the end of Vol. III. By Dr. PIERRE JANET, Professeur de Psychologie au Collège de France. Paris: Félix Alcan. 1919.

In these three volumes, Professor Pierre Janet has attempted to put together the nature and action of the various means that have been used at

different periods in the world's history for combating the psychoneuroses. He first deals with "Moral Action." This he divides into miraculous cures under the influence of religion, magic or animal magnetism; then he takes philosophic treatment such as Christian Science, and finally what he calls "Moralisation médicale," of which he gives as his example treatment by Dr. Dubois of Berne.

The second part of the first volume is devoted to the forms in which automatic action is used to produce a cure. Here, he deals with the whole history of hypnotism and suggestion. This is a most valuable summary of the forms employed at various times and the aims of the different schools of thought.

The second volume is devoted to treatment by rest, by isolation, and by what Janet calls "Liquidation morale." As this comprises psycho-analysis and Freud's views of the nature of these disorders, it will be obvious that the term "psychological economy" has been made to cover an unusually wide ground.

The third volume is devoted to what the author calls "Acquisitions psychologiques" such as re-education, the production of crises and somnabulisms, the treatment by excitation and moral direction.

It is obviously impossible to give any idea of the contents of this work in a short space. It is a book that must be studied carefully, and is full of information with regard to the various forms of treatment that have been adopted from time to time. It is, however, doubtful whether the author was wise to divide the various sections in so logical a manner; for many of the views that have been held at different periods do not fall into their proper position, unless they are treated in sequence; for example, it is important to remember the part played by Janet's own work in the evolution of Freud's views, and subsequently those held by Jung and others.

The third volume is a valuable index of authors cited and of the subjects treated throughout the work.

Functional Nerve Disease. Edited by H. Crichton Miller, M.A., M.D. Pp. 208. London: Henry Frowde, Hodder and Stoughton, 1920.

Dr. Crichton Miller, as general editor, has gathered together short contributions from ten authors on the subject of functional nervous disorders. The book is divided into four main portions; these deal with the physical, hysterical and anxiety factor, followed by two chapters on the management of the neurotic, and a summary by Dr. McDougal. The various chapters are in reality independent of one another, for each author is permitted to hold his own views, and does not hesitate to criticize the different schools to which his colleagues belong. For those readers who are already familiar with the present trend of psychopathology, it is of great interest to have the various views presented with such clearness. Each chapter is prefaced by a synopsis which enables the reader to appreciate the tendency of the ideas put forward by the writer; were this not the case, the concentrated writing of some of the chapters would be somewhat difficult to understand.

Treatment of the Neuroses. By ERNEST JONES, M.D. Pp. 233. London: Baillière, Tindall and Cox, 1920.

This is an interesting little book written with all the ease and clearness we have grown accustomed to expect of this author. Although it is supposed to be concerned with the treatment of the neuroses, the main portion of the book is occupied with hysteria, and the remaining functional disorders are treated comparatively shortly. This is, however, justified by the fact that when discussing hysteria and the various views that have been held as to its nature, Dr. Jones deals with most of the processes which underlie the general neuroses. To whatever school the reader may belong, this book can be strongly recommended as an introduction to modern views on this subject.

Psychoneuroses of War and Peace. By MILLAIS CULPIN, M.D., F.R.C.S. Pp. 127. Cambridge: University Press, 1920.

Every writer on the psychoneuroses who has had any extensive personal experience, must of necessity adopt an individual attitude towards these disorders. Dr. Culpin worked originally as a surgeon, and was struck with the large number of cases in which psycho-pathological states were mistaken for surgical or medical conditions. Finally, towards the end of the war, he obtained an opportunity of working under more favourable conditions in an institution devoted to the neuro-psychoses; this monograph is the result of his final experience. It is somewhat loosely written, but is full of interesting material. The way in which the cases are strung together in the text makes it somewhat difficult to read for the practitioner who is not familiar with the conditions described. But it is of great interest to those who have followed the recent trend towards the recognition of the psychical element which forms so important a part in what appear to be straightforward surgical or medical cases.

A Manual of Neurasthenia. By IVO GEIKIE COBB, M.D. Pp. 366. London: Baillière, Tindall and Cox, 1920.

This is a systematic attempt to construct clinical signs and symptoms which accompany what is called neurasthenia in its widest sense. The mental aspects are treated shortly, and then various symptoms such as headache, gastro-intestinal disturbance, sensory disorders, insomnia, and what are called "objective signs" of neurasthenia are dealt with seriatim. The second part of the book deals with treatment, consisting of general hygiene, diet, the use of drugs, climate and electricity: psycho-therapeutic treatment occupies thirteen pages only.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I to XXIII inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

[November, 1920.]

BRAIN.

PART 3, VOL. 43.

ON THE SYMPTOM-COMPLEXES OF LETHARGIC ENCEPHALITIS WITH SPECIAL REFERENCE TO INVOLUNTARY MUSCULAR CONTRACTIONS.

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I.—Introduction.

When a disease, particularly one involving the central nervous system, is isolated in the first place by clinical observation, it follows of necessity that only cases corresponding to a single symptom-complex can be identified as belonging to it. With increasing knowledge the originally clear-out conception of the malady becomes inadequate to include the inevitable variations from type that are observed. It has to be broadened and it loses the artificial precision of its earliest form.

Investigations into the ætiology and morbid anatomy of the malady proceed simultaneously with the extension of knowledge on the clinical side, and, finally, it becomes possible with reasonable accuracy to describe a clinical picture characteristic of the disease and to establish its main clinical variations and forms. It may be possible in some cases to correlate these with variations in the intensity or distribution of the underlying lesions in the nervous system.

It is clear that during this phase of growing experience much modification and revision of opinion as to the significance and value of observed phenomena may be necessary.

In the case of lethargic encephalitis we are still in this phase of growing knowledge. We know little of its ætiology and the full range of its clinical manifestations has probably not yet been determined. In the absence of any bacteriological or serological standards it is not always possible to decide whether a given symptom-complex should be included in the rapidly growing group attributed to this disease or not. It seems probable from a study of the literature that from time to time cases are incorrectly included, while others that belong to the group may equally well be rejected from lack of these standards.

The original clinical picture by which, under the name of botulism, the disease was first diagnosed in this country, soon gave place to various forms or types, the number of which is steadily on the increase. As Buzzard and Greenfield [5] have pointed out, it is to be expected that an inflammatory disease of the nervous system should give rise to a versatile symptomatology ; the structure of this system is so complex and the functions of its several parts so highly specialized. Indeed, it is easy to conceive of innumerable possible combinations of symptoms—that is of possible syndromes—we might reasonably expect to encounter, though it would not be correct to call them all "types."

Many schemes of classification for the various syndromes of lethargic encephalitis are to be found in the literature. Most of them are purely symptomatic ; some prominent symptom in a series of cases being chosen as the basis for the definition of a type. Other writers employ mixed anatomical and symptomatic groupings. The result has been that each case either simultaneously or at some stage of its course falls into almost as many groups as it has symptoms. The recently named "myoclonic encephalitis" affords a good example of this. Rhythmical clonic contractions and fibrillary twitchings of muscles, or of parts of muscles, have been recorded in lethargic encephalitis by numerous observers during the past two years. In the recrudescence of the disease during the past winter (1919-20) they have been a remarkably prominent feature. In some recently reported cases they have dominated the clinical picture entirely (Ellis, [10]), while in a few instances (Sicard and Kudelski [17, 18, 19]) clonic muscular contractions have been the only somatic nervous symptom observed. At first sight it might seem justifiable, with Sicard, to regard so clear-cut and striking a symptom-complex as a new and distinct type of the disease. However, experience indicates that the particular combination of symptoms recorded by Sicard is extremely rare in a pure form ; that usually it occurs either

superimposed upon, preceding, or following a whole series of other symptoms in any given case; and that clonic muscular contractions occur as a symptom in cases of every clinical variety. Nevertheless, following Sicard, numerous writers now group as "myoclonic encephalitis" cases differing in the widest possible manner and often having nothing in common clinically except clonic twitchings of one or more muscles or parts of muscles. Moreover, the term "myoclonic" has been applied to such a variety of involuntary muscular contractions and movements by French writers that it has come to have little or no meaning. The unsatisfactory nature of classification of this kind is exemplified by such a case as that reported by Bourges and Marcandier [4] as a "forme mixte à symptômes intriqués (co-existence de somnolence, paralysies partielles oculaires, mouvements choréo-athétosique, secousses myocloniques, catatonie et troubles délirants"). While Kahn reports [12], "Un cas d'encéphalite successivement névralgique, psychique, choréiforme, myoclonique, léthargique avec séquelles hémi-myoclonique après la guérison."

Such complex syndromes are by no means exceptional and the disease which can produce them clearly defies symptomatic classification.

Quite possibly no permanent classification can be devised in the present incomplete state of knowledge, but it is reasonable to suppose that an analysis of the features of the disease might reveal some fundamental character upon which a uniform scheme of description could be founded.

The symptoms of lethargic encephalitis fall naturally into the following groups:—

General Symptoms—Signs of toxic infection ..			Fever. Shivering. Cutaneous eruptions. Pains and malaise. Headache. Gastro-intestinal disorders, such as vomiting, constipation, foul tongue, &c.
Nervous Symptoms	General	"Positive"	Delirium, mania, restlessness, hallucinations, &c.
		"Negative" ..	Lethargy, coma.
	Focal ..	"Positive"	Convulsions. Involuntary movements and muscular contractions Ataxy, rigidity. Pains.
		"Negative" ..	Paralyses.

The term "positive" and "negative" are employed in the sense in which Hughlings Jackson originally used them—"positive" symptoms being expressions of exalted function either from irritation or from loss of higher control, and "negative" symptoms expressing depression or loss of function from destruction of nervous structures or otherwise. A recognition of these two classes of symptoms is essential to a comprehension of any nervous disease.

Not all these symptoms are represented in a single case, nor are those present of equal value in every instance. Thus, the general symptoms of toxic infection are minimal or entirely escape observation in some instances, while in others they dominate the clinical picture at the onset or even throughout the illness.

If ten consecutive cases were taken and considered in the light of the scheme given above, it is quite possible that not a single symptom would be common to them all. If we seek for a common factor we shall find it only in the presence of focal or localizing nervous symptoms.

Among those symptoms that are more definitely focal in origin, the localization of the positive motor phenomena presents considerable difficulty, and very diverse views have been expressed as to their cause and site of origin. Therefore, as they may be the only focal symptoms present in a given case, an attempt to analyse their nature and cause is an essential preliminary to any sound basis of description or classification. This question will be considered later, but in the meantime a few general observations on the symptomatology of lethargic encephalitis may be made.

At the time of its first recognition, the disease was remarkably "negative" in the character of its symptoms: lethargy and paralyses of certain motor cranial nerves made up its clinical picture in most instances, and although certain "positive" general and focal nervous manifestations were recorded from 1918 onwards, they were certainly not so characteristic of the malady as they have since become. Doubtless, there are many factors responsible for this apparent change in the clinical aspect of lethargic encephalitis during the past few months. It is very easy to exaggerate the importance of this change. Undoubtedly a widened conception of the disease and the admission of cases that could not have been recognized earlier as belonging to it have been contributory causes. But it also must be remembered that although overlaid by more obtrusive positive symptoms, the same negative symptoms that attracted attention to the disease originally still occur with considerable frequency. As Netter [15] has pointed

out in this connection, it has long been known that a poison may cause either exaltation or depression of function, at one time producing convulsions, at another paralysis, as alcohol, lead, and the virus of rabies are well known to do. Further, the clinical variations of an infective disease in successive epidemics are quite familiar, and although there certainly is some evidence of more extensive involvement of the nervous system now than in the earlier cases, yet we need not regard a change in the localization of the virus as even the most important of the many factors concerned.

Whatever their cause may be, the frequency and variety of "positive" symptoms, both general and focal, have increased the clinical diversity of lethargic encephalitis still further, so that a comprehensive view of its essential features is becoming increasingly difficult.

Nevertheless, an appreciation of the fact that the same principles govern the localizing of "positive" as of "negative" symptoms, of a clonic contraction as of a palsy, renders the problem less complex than appears at first sight and makes the prospect of finding a satisfactory uniform basis of description more hopeful.

A consideration of *acute poliomyelitis* in the light of the scheme given above offers many interesting points of contrast between that disease and lethargic encephalitis. In the former the general symptoms are far more constant in form and duration than in lethargic encephalitis. They always precede the onset of paralysis and rarely last more than four or five days. When they abate the activity of the virus may be considered to be spent. In lethargic encephalitis, on the other hand, as we have seen, they vary greatly in intensity, form and duration, and, as Netter [15] has suggested, it is probable that the virus remains active for weeks or months. The nervous symptomatology of poliomyelitis is characteristically "negative" and predominantly focal in type. Yet even in this disease, when the cerebrum is involved, convulsions may occur. In this connection a case described by Leonard Parsons and quoted by Batten [3] as one of poliomyelitis is of considerable interest at the moment. It was that of a child of 2, who was suddenly taken ill with fever, a fit and sleepiness. The fits recurred next day; the child regained consciousness but remained for three weeks "apparently conscious, took her milk, but her eyes were fixed and staring, and she did not appear to see anything nor did she speak." For three weeks her limbs showed a slow rhythmic tremor and were rigid. There was no paresis nor any change in the

reflexes. Recovery ensued after five weeks. In 1913 this case was diagnosed as poliomyelitis, it is doubtful if it would be so called now. With rare and questionable exceptions such as this, it may be said that the nervous symptoms of poliomyelitis are negative, or paralytic, and do not show the selective and restricted character of the palsies seen in lethargic encephalitis, which commonly involve certain motor cranial nerves only. For the long-continued fibrillary and clonic contractions of muscles seen in the latter disease there is no parallel in poliomyelitis.

In poliomyelitis classification and description have an anatomical basis. The disease is much less polymorphic than lethargic encephalitis, and its focal symptoms are readily interpreted. Yet even here Wickmann found it necessary to add an "abortive" group of cases to his purely anatomical grouping. There is no reason why in the case of lethargic encephalitis a similar uniform basis of description should not be attempted, though, undoubtedly, the problem is far more complex. Nevertheless, nothing but good can come from the abandonment of the numerous symptomatic classifications that serve only to obscure our outlook on the disease.

II.—Clinical Cases.

In this section are briefly described seven cases which were observed successively in the practice of University College Hospital. Five of them were under the care of the Medical unit, and for permission to record the remaining two I am indebted to the courtesy of Sir John Rose Bradford (Case 1) and Dr. F. J. Poynton (Case 5).

These cases were not selected, and their clinical diversity is not, therefore, artificial, but represents the disease as it has been observed in the practice of a single hospital during the past few months, though the cases are not recorded in the order in which they were seen.

At the head of each case the symptoms are summarized according to the scheme given in the first section of this paper.

Case 1.—*Lethargy and Signs of a Cortical Lesion.*

General Symptoms.—Fever, breathlessness on exertion, anorexia, constipation, foul tongue, congested fauces, headache and diffuse pains.

Nervous Symptoms. — (General) Lethargy passing into coma, general muscular hypotonus and diminished tendon reflexes. (Focal) Right hemiparesis and Jacksonian fits.

Alice R., married, aged 42. On January 12, 1920, she suddenly came over

faint and giddy while at housework. She lay on her bed and went to sleep. During the following week she complained of headache, lassitude, vague, fleeting pains in limbs and body, and increasing drowsiness, so that at times she could hardly keep awake. She was very breathless on exertion. For about a week she kept at her work, and her husband appeared to notice nothing abnormal in her. She had previously enjoyed excellent health.

On January 19, 1920, she suddenly became very much worse; she noticed that she was weak on the right side, and that her right arm and leg were twitching.

On examination on the following day she was very somnolent and slept unless aroused. This was easily done, however, and she could then give a rational account of her illness. Temperature was 103° F., pulse 132, and respirations 32. Her tongue and lips were dry and coated and the fauces congested. The thoracic and abdominal viscera appeared healthy. The urine was normal. She complained of headache and of pain in the right side of the chest.

At increasingly frequent intervals, until her death on February 9, 1920, there were right-sided Jacksonian fits of face, arm, hand and leg. The clonic jerkings began in the thumb and index-finger and spread up the arm. The lower part of the face on that side and the leg were simultaneously involved. The movements, which occurred at the rate of about three in two seconds, were most forceful and enduring in the hand and forearm; the leg was least affected. In the upper limb the flexor group seemed most affected; in the leg only the limb extensors and plantar flexors could be felt to contract. Associated with this condition was a progressive right hemiparesis, in order of severity, hand, arm, face, leg. The muscles were flaccid. All tendon jerks were sluggish, right and left equally. No abdominal reflexes were obtained, and the right plantar response was extensor, the left normal. As far as could be determined there was no disturbance of cutaneous sensibility; other forms could not be tested. There was incontinence of urine and fæces.

The patient gradually became comatose, broncho-pneumonia developed, and she died four weeks from the onset of the illness. At no time were there delirium, pain, or ocular paralyses. There was no increase of lymphocytes in the cerebrospinal fluid.

At post-mortem the soft meninges appeared congested, but no other abnormality was seen. The precentral and post-central gyri and the basal ganglia of both sides, the cerebellum, midbrain, and medulla were examined microscopically. In the cerebral cortex and the basal ganglia there was considerable round-celled infiltration of the perivascular spaces, but no other lesion was noted. No difference could be detected between the two sides. The cerebellum and brain-stem were normal.

In this case the focal symptoms indicated a combined irritative and destructive lesion of the cerebral cortex; the paresis and the convulsions having the same localizing value.

Case 2.—Lethargy and Signs of Basal Ganglia and Tegmental Lesions.

General symptoms.—Constipation, fleeting limb pains.

Nervous symptoms.—(General) Lethargy. (Focal) Defective upward movement of eyes, nystagmus, mask-like face, rigidity and tremor of left limbs.

Harry T., aged 14. His mother stated that five weeks before his admission to hospital he came home from work on a Saturday afternoon complaining of being very sleepy. He lay on his bed and slept with intervals, when he was waked for meals, until Monday morning, when his drowsiness was still unabated. During the succeeding five weeks he went to sleep whenever he was left to himself, but was always easy to rouse. He ate his meals fairly well and seemed "right in his mind," though his mother noticed that he "grinned silly" at her when spoken to, and that his left arm and hand shook and were clumsy when in use. He was very constipated, and complained from time to time of vague pains in his limbs. These were never severe.

On examination (July 5, 1920) he lay in bed apparently asleep. He awoke and became quite alert when addressed and could answer simple questions readily and accurately. He displayed a complete loss of initiative, and neither moved nor spoke unless made to do so. His face was vacant and expressionless, but periodically a slow smile dawned on his face, spread, and remained fixed often for as long as a minute before it faded slowly away. Articulation was normal, and he could swallow normally. Vision and hearing normal.

The pupils were normal. Upward movement of the eyes was distinctly limited in range. Lateral movements were normal in range and association. On fixation, particularly on lateral deviation, a coarse, rapid nystagmus appeared. This was better sustained and more rapid on looking to the left than on looking to the right. No vertical nystagmus, no squint or diplopia. No ptosis.

There was moderate paresis of both halves of the face, especially of the upper part; thus the frontales were extremely weak. In movements carried out to order the right half of the face acted more rapidly and amply, but on smiling the left half "took up" much sooner and acted better than the right. The other cranial nerves showed no defect.

Motor system: The left arm and hand were held flexed and abducted, but otherwise his general attitude was in no way peculiar. All mass movements were carried out very slowly as though he were stiff or sore. He moved and walked just like a case of paralysis agitans, and retropulsion was easily induced by making him hold back his head or by a slight push. The left arm and leg showed a curious "sticky" rigidity like that seen in paralysis agitans, and clearly differing from that seen in lesions of the pyramidal system. Movements of the left hand and digits were very slow and clumsy and a fine tremor of the forearm and fingers appeared on movement. The leg also was slow in movement but there was no tremor. Isolated movements of the right limbs were carried out with normal facility and speed, but in mass movements the pace seemed to be set by the capacity of the affected left limbs.

There was no weakness of the left limbs, power and muscular development were normal throughout. The tendon jerks were of normal facility and equal on the two sides. The abdominal reflexes were all present and the plantar reflexes flexor in type. Sensation was normal and there was no pain. Sphincter control was normal.

There was no fever. The cerebrospinal fluid was normal in every respect, there was no lymphocytosis or increase of globulin. Wassermann negative.

During the following four weeks the condition altered very little, but the lethargy gradually lessened.

At present (September 1, 1920) there is no lethargy, the rigidity and slowness of movement of the left arm are improved, but general movements, the defect of eye movements, the facies and nystagmus are unaltered.

In this case the focal symptoms indicate a lesion of the basal ganglia and the tegmental region of the midbrain. The unilateral character of certain of the symptoms is unusual, but in other respects they are of a kind quite familiar and may be regarded as examples of a mechanism released by defective activity of co-operative centres.

Case 3.—Delirium and Extreme Restlessness (Choreiform) followed by Lethargy. Signs of Midbrain (peri-aqueductal) Lesion.

General symptoms.—Fever, breathlessness on exertion, anorexia, constipation, foul dry tongue and lips, morbilliform eruption and joint pains.

Nervous symptoms.—(General) Insomnia, nocturnal delirium, hallucinations, extreme restlessness resembling severe chorea. All passing suddenly into profound lethargy after thirty-six hours. General muscular hypotonus and diminished tendon jerks. (Focal) Third, fourth, sixth and seventh nerve palsies.

Louisa F., aged 14. On May 24, 1920, the patient's mother noticed that she was very short of breath on exertion. Two nights later she began to be sleepless and restless at night. This became nightly worse, so that on the night before admission she was violent, could scarcely be kept in bed, threw herself about, appeared to be having terrifying visions and shouted that men were after her. At the same time "a rash suddenly came out on her like measles." During this period of onset she was constipated, had no appetite, complained of severe pain in the right wrist which made her cry out at times, and she seemed feverish. Admitted to hospital as a case of chorea.

On admission (May 31, 1920): She lies on her back in bed, continuously tossing about. She flings her head and limbs about with great force and has to be kept from falling out of bed by a strong sheet. The condition resembles severe adult chorea. She is flushed, her skin is hot and dry, her lips are dry and cracked and her tongue coated. Temperature 102° F.

Over legs, elbows, buttocks and shoulders there is a bright red papular rash. At the apex of the papules are small dark red crusts like dried hæmorrhages.

Although this is most marked over areas where friction is greatest it is also present on protected areas of skin and cannot be entirely due to rubbing.

Nervous system: She is in a muttering stupor when not disturbed, but when spoken to half opens her eyes and gives rational replies to simple questions, though she can give no account of her illness. Her answers are perfectly articulated.

Vision and hearing appear unaffected. She does not complain of tinnitus, but says that she sees double.

Cranial nerves.—Her pupils are equal, central and circular. They react fairly well to both light and accommodation. There is marked double ptosis with feeble overaction of the frontales when she opens her eyes. The eyes do not move at all parallel, but no single muscle can be incriminated and all movements appear limited in range. There is no nystagmus. There is diplopia. There is profound weakness of both sides of the face. She can close her eyes, but there is scarcely any movement round the mouth. Her tongue is protruded straight and well and can be maintained steadily protruded. All its movements are perfectly normal. The motor and sensory fifth nerves are normal. Articulation, deglutition and phonation are normal.

Motor system.—There is general hypotonus and some general weakness, but no true paresis, nor localized weakness. The constant movements of head, trunk and limbs closely resemble those of chorea, but there appears some evidence of voluntary control when carrying out movements to order. The steadiness of the tongue moreover is unusual in severe chorea. These two facts and the general character of the movements suggest that although the resemblance to chorea is striking it is mainly superficial and the presence of the cranial nerve palsies confirms this view. The tendon jerks are all sluggish, but obtainable. They are equal on the two sides. Abdominal reflexes doubtful. Plantars flexor. There is no demonstrable defect of sensation. She complains of pain in the region of the right wrist, but no local abnormality of any kind can be detected.

(June 2, 1920.) After eighteen hours the movements ceased suddenly and the patient subsided into a profound lethargy. She now resembles the ordinary form in which lethargy and signs of a peri-aqueductal lesion co-exist. Temperature remains raised. She can be aroused for feeding and to answer questions. The pain in the right wrist has gone.

June 15, 1920. During the past two weeks the cranial nerves have rapidly regained normal function. There is now very slight ptosis. The eye movements are normal and there is no facial weakness. She is still somewhat lethargic and sleeps most of the day. At night she is more wakeful and complains of pain in the region of the left shoulder. This pain is localized in distribution and not neuralgic in character. There is no local condition to account for it. The rash has disappeared and the general condition is good.

On admission the cerebrospinal fluid was examined. There were 160 lymphocytes to the cubic millimetre. There was no leucocytosis in the blood.

The extreme restlessness accompanying the delirium when the patient first came under observation closely resembled severe chorea, and it was with this diagnosis that she was admitted to hospital. The presence of cranial nerve palsies, the sudden onset, and certain minor features which are emphasized in the foregoing account sufficed for the recognition of lethargic encephalitis.

The extreme violence of the movements, their incessant and disorderly character and their association with marked psychical excitement make it extremely probable that we are dealing here with an irritative lesion of higher cerebral centres, rather than any release mechanism.

Case 4.—Lethargy and Acute Cerebellar Ataxy.

General symptoms.—None observed.

Nervous symptoms.—(General) Acute onset of headache, vertigo and vomiting. (Focal) Acute cerebellar ataxy.

Captain F. C., aged 27. On May 5, 1920, after a hearty breakfast, and having been previously in apparently excellent health, he was suddenly seized with severe headache, giddiness and vomiting. These symptoms persisted throughout the day. He was examined during that evening and then lay curled up on his bed on his right side. He showed extreme photophobia, but was not irritable. He was fully conscious and rational, but could not give a good account of his symptoms on account of a gross defect of articulation. His friends stated that he had "stammered badly" all that day, though he had never been known to do so before. There was no fever, pulse rate was 64, and respiration rate 20.

Re-examined in hospital on May 7, 1920. His general condition was improved, the vomiting had ceased and the headache diminished, though some occipital pain remained and was aggravated by movement. There was no sense of giddiness. He was distinctly lethargic and lay curled up in bed and apparently asleep until spoken to, when he became quite alert and rational.

There was extreme ataxy of articulation, which was slow and accompanied by overaction of the facial muscles.

Vision: acuity, fields and fundi, normal. Hearing normal, some tinnitus. The pupils were equal and reacted normally. There was no ptosis, or diplopia, but there was a slight divergent squint of the right eye, probably congenital. On lateral deviation a fine rapid nystagmus appeared.

There was a doubtful paresis of the lower part of the left side of the face. Deglutition and phonation were normal. The tongue was protruded normally.

There was gross ataxy of the arms of cerebellar type, the right arm was particularly unsteady and jerky on movement. He walked with a wide base and had to be supported. There was no tendency to fall in any constant direction. The tendon reflexes were brisk and equal on both sides, the

abdominal reflexes were present. The left plantar reflex was doubtfully extensor in type. There was no discoverable defect of sensation.

The cerebrospinal fluid was normal in cell and globulin content. The Wassermann reaction was negative. There was no leucocytosis in the blood.

During the six weeks after admission all these symptoms gradually cleared up and on discharge in June slight ataxy of speech, which was still somewhat staccato in character, and some intention tremor of the right hand and arm, were the only noticeable symptoms. When fatigued some unsteadiness of gait appeared.

In this case the focal symptoms are those known to be associated with a negative or defective lesion of the cerebellum. They are in no way peculiar to lethargic encephalitis.

Case 5.—Lethargy and Signs of Irritation of Lower Motor Neurones.

General symptoms.—Lassitude, anorexia and constipation.

Nervous symptoms.—(General) Insomnia, nocturnal delirium, hallucinations passing into somnolence. General muscular hypotonus and diminished tendon jerks. (Focal) Rhythmic contractions of certain arm and abdominal muscles.

George C., aged 44. (History obtained from friends.) Had a "chill" last April. Since this he has not been well. He has complained of being tired, without appetite, and constipated. On May 26 he began to be sleepless at night and to mutter in his sleep. During the following week he complained of severe pains in his limbs, was told by his doctor that he had rheumatism and advised to stop work. Early in June he had severe pain in the chest and was thought to have pleurisy. Since the first week in June he has been confined to bed, semi-unconscious during the day and restless and violent at night. He appeared to be having terrifying visions, talked excitedly and had to be kept in bed by force. He was incontinent of urine and very constipated.

On admission to hospital (June 19, 1920) he was profoundly lethargic, but could just be roused to answer simple questions. He could give no account of his illness. He understood spoken speech perfectly and replied in whispers. He complained of no pain. He passed urine in the bed. Temperature was 101° F., pulse 128, and respirations 32.

Hearing and vision appeared normal. Fundi were normal. The cranial nerves showed no defect. There was general muscular weakness and hypotonus, but no localized paresis. There was no spontaneous tremor or muscular contraction. No sensory loss could be detected. The tendon jerks were all very sluggish, the abdominal reflexes not obtainable and the plantars flexor.

The cerebrospinal fluid was normal in every respect, and there was no leucocytosis in the blood.

During the following two months his general condition and his lethargy remained unchanged and he became slowly thinner. It was found during this period that on passive manipulation of the arms, and after such forceful voluntary contractions of the muscles of these limbs as he could be induced to

carry out, there appeared slow clonic contractions of certain forearm and small hand muscles. The rate of these was fairly constant and was about 48 per minute. These contractions which involved the whole muscle appeared first in the supinator longus, then in the extensor group of muscles, and finally in the thenar muscles. They were feeble, and after two or three minutes they died away until the limb was further manipulated.

Similarly, abdominal palpation evoked feeble rhythmic contractions of the upper segments of the recti on both sides, which caused the umbilicus to be drawn upwards at each twitch. Very occasionally the right sternomastoid was seen to twitch spontaneously in the same manner and at the same rate.

September 1, 1920. The patient's general condition is gradually failing, he is losing weight and a large sacral bedsore has appeared. The lethargy remains unchanged and he can still be aroused and can carry out simple orders and answer simple questions in a whisper.

The rhythmic contractions described above have undergone an interesting change. Passive stretching of the forearm and hand muscles now evokes fibrillation and occasionally fascicular twitching of large strands of muscle. If passive manipulations be maintained for several seconds a slow rhythmic contraction, very feeble and producing very little movement of the digits, of the flexor muscles in the forearm appears and lasts for some seconds. Neither fibrillation nor contraction can be induced in the upper arm or the lower limb muscles, nor in the recti abdominis.

The only somatic nervous symptoms observed in this case were fibrillary and fascicular twitching and rhythmic contractions of certain muscles. The distribution of the affected muscles was quite random, and there was no evidence of synergically associated muscles being affected. Moreover, the contractions were for the most part not spontaneous, but induced by passive manipulation of the muscles, and it seemed that as the tendency to "myoclonus" abated fibrillary and fascicular twitching took its place.

The random distribution of the clonic contractions in muscles not functionally associated, its restriction in certain circumstances to segments, strands, or even smaller muscle-fibre bundles (fascicular and fibrillary twitching), indicate very definitely that we are in all probability not dealing with a lesion of neurones of high physiological levels, but with an affection of the lower motor neurones, presumably irritative in character.

Case 6.—Delirium, Confusional State, Restlessness and Pain followed by Signs of Irritation of Lower Motor Neurones.

General symptoms.—"Influenza" followed by persistence of pains in back and limbs, fever, swelling of ankles, albuminuria and constipation.

Nervous symptoms.—(General) Headache, insomnia, restlessness, delirium

and confusional state, general muscular weakness and hypotonus. (Focal) Rhythmic contractions of isolated muscles.

Gertrude B., married, aged 38. In March, 1920, patient had an illness, of which no details are available, which was diagnosed as influenza. Following this she did not recover completely, but complained of lassitude, fleeting limb and back pains, some fever at night (100°), swelling of the ankles and sleeplessness.

When seen on April 5, 1920, nothing abnormal was noted beyond œdema of the ankles and marked albuminuria.

On April 10, 1920, she became "light-headed," and complained of severe pain in the right arm, epigastrium and back.

On April 12, 1920, an erythematous rash appeared on abdomen, back and arms, with forcible twitching of muscles of neck, upper limbs and shoulders. This was accompanied by severe pain in the affected muscles. The temperature was 100° F.

On April 14, 1920, she was less restless and excited, but was very confused as to her whereabouts and did not recognize her relatives.

On April 17, 1920, the albuminuria had disappeared, the temperature remained slightly raised. The rhythmic muscular contractions persisted and she was restless and delirious at night.

She was admitted to hospital on May 31, 1920. She was pale and generally wasted and had a harassed, anxious expression. There was no trace of lethargy. She was quite disoriented as to time and place, and identified those in charge of her with various members of her family. She was inclined to be talkative and gave extravagant accounts of her doings during the day. Her mental state was like that seen in Korsakow's psychosis. Speech was normal. Hearing and vision were normal.

The pupil and their reactions were normal, the ocular movements were normal. There was no ptosis, squint, diplopia or nystagmus. All the cranial nerves were normal,

There was general wasting, weakness and flaccidity of the muscles, but no localized paresis. All movements were unsteady, probably on account of the extreme general weakness. There was no tremor. The weakness was so marked that she was very helpless and could do nothing for herself. No sensory change could be detected. The tendon jerks were all brisk, the abdominal reflexes were present and equal on the two sides. The plantar reflexes were doubtfully extensor on both sides. There was no sphincter defect.

Examination of the abdominal wall revealed a constant rhythmic contraction of the lower segments of the recti abdominis, particularly on the right side. This caused a rhythmic downward movement of the umbilicus (downwards and to right). At times, usually while she was asleep, the left sternomastoid showed a similar clonic contraction. The rate of the beats in both instances was 48 per minute. The abdominal muscle twitching undoubtedly increased in force when the muscles were palpated, and even when the patient was being

examined and was consciously under observation, but it continued during sleep. It was noteworthy that if the abdominal reflexes were rhythmically stimulated at a faster rate than that of the spontaneous twitching, the rhythmic reflex responses completely replaced the spontaneous beats for the time being; that is, both movements did not occur simultaneously, one or the other monopolized the muscle.

The cerebrospinal fluid at the time of admission was clear and free from excess of cells (average of two cells per c.mm. in several counts).

The patient remained under observation for two months. During this time her mental state gradually became normal. There was at no time either lethargy or cranial nerve palsies. Her general condition steadily improved and she regained strength and weight.

The abdominal muscular twitchings became less forceful, but persisted throughout and were still present when she left hospital at the end of July.

In this case, as in Case 5, the sole somatic nervous symptoms indicated an irritative lesion of lower motor neurones. The considerations advanced in the previous instance apply here and indicate that we are dealing with involuntary muscular contractions differing in nature and site of origin from the clonic convulsive movements seen in Case 1.

Case 7.—Signs of Irritation of Sensory and Motor Spinal Roots.

General symptoms.—None observed or recorded in history.

Nervous symptoms.—(General) None observed or recorded in history. (Focal) Intense neuralgic pain in distribution of cervical sensory roots, followed by the appearance of persistent fibrillary twitching in muscles of right shoulder girdle and arm.

Charlotte M., married, aged 36. On about June 20, 1920, having gone to bed feeling perfectly well, she awoke at 2 a.m. with intense pain "in the spine of the neck" running up to the back of the head. The muscles at the back of the neck "seemed to be all on the work" and her head was drawn backwards. So severe was the pain that she had to cry out aloud.

In the morning the pain had spread to the right shoulder and seemed settled on the right side of the neck also. It was still intense and seemed to shoot outwards from the back of the neck down on to the shoulder and the upper part of the right side of the chest. She noticed that all the muscles round the right shoulder, under the right breast and in the arm as far as the elbow "were all on the twitch." Her friends "could see them wriggling through her blouse." These twitchings did not move the limb at all, but the muscles looked "as though there were worms wriggling beneath the skin." During the day the "wriggling" spread to the muscles of the forearm, and the whole limb became weak, so that she could not lift her arm up to her mouth, and her grasp became unsteady and weak. The limb felt very heavy. This

condition persisted for two days and then became very much less marked, though even yet when she uses the arm they start again round the shoulder. The right arm has remained weak, she cannot hold a pen or a needle partly on this account and partly because the hand is unsteady and shaky.

The neuralgic pains persisted unabated for two weeks. During this time she could not rest for them, she slept hardly at all, and had to walk about constantly with the right arm supported to ease the pain. For the past three weeks the pain has gradually diminished, though even yet she cannot lie on the right shoulder without bringing it and the twitchings on again.

There was no lethargy at any time. There is no history of cranial nerve palsies, nor of any constitutional disturbances, such as fever, headache, constipation, nausea or vomiting.

She states that she has always been highly strung. This has been much worse since the air raids, which have made her very nervous and easily upset. Her general health has always been good. She has had three pregnancies and three healthy children born at full term. Two died during the first year of life from "convulsions," and the third is alive and well.

On admission to hospital (July 26, 1920) she was found to be a well-nourished woman of healthy appearance. Thoracic and abdominal viscera healthy. Intelligence and emotional tone normal. Vision and hearing were normal. There was no defect of cranial nerves.

The motor system: There was weakness and extreme tenderness to pressure of the following muscles of the right shoulder girdle and arm: sternomastoid, trapezius, all the scapular muscles, pectoralis major, deltoid, the rhomboids, triceps, biceps. The forearm and small hand muscles were slightly tender. There was some tenderness of serratus magnus and distinct winging of the scapula. There was general hypotonus of the affected muscles. All movements of the limb were limited in range on account of weakness, and active movements produced a tremulousness of the hand and arm that was partly due to this weakness.

Active or even passive movements of the various segments of the limb produced at once ample fascicular and fibrillary twitching of the muscles involved, and the contractions partook of the characters of both fibrillation and clonus. For although the twitchings were regular in frequency, they involved varying proportions of a muscle in different contractions, so that contractions of small muscle bundles, of strands of muscle running the whole length of the muscle and of the whole muscle simultaneously were seen. These contractions did not cause actual movement of segments of the limb.

There was no other abnormality of the musculature elsewhere in limbs or trunk.

Over the upper arm and fore-quarter on the right side there was a qualitative alteration of cutaneous sensation. All touches were felt, and pinprick was painful, but sensations appeared diffuse and not accurately localized and differed in quality from normal areas of skin. This area could not be accurately mapped out.

The tendon jerks in both arms were present and no definite inequality between the two sides could be made out. The knee and ankle jerks were normal, the abdominal reflexes present and the plantars flexor in type.

The patient was only under observation on two occasions during the last week in July and her present condition is not known.

This case did not come under observation till convalescence was well established, but nevertheless the physical signs were in accord with the history of the early stages of the illness. They were those of an irritative lesion of cervical spinal roots, both motor and sensory. If this view of the localization and character of the lesion be correct, it confirms the hypothesis that the "myoclonic" and fibrillary twitchings recorded in the two previous cases are dependent upon irritation of the lower motor neurones. In these the absence of definite tenderness of the twitching muscles and of neuralgic root pains suggests that the lesion was in grey matter, rather than in the roots.

Sicard [20] records an exactly similar case.

III.—Involuntary Movements and Muscular Contractions; the so-called "Myoclonic" and "Choreiform" Manifestations of Lethargic Encephalitis considered as Irritative Phenomena. The Contrast between the Paralytic and the Exciting Action of the Virus.

The general impression derived from the study of this small series of cases accords with that obtained from a review of the literature of lethargic encephalitis: namely, that the constitution of the symptom-complex in any given case appears to be largely fortuitous and to follow no laws that we can yet determine. Any and every part of the nervous system may be involved and there is no combination of symptoms that we may not encounter. In consequence, many cases cannot be placed in any type unless types are to be multiplied until they cease to have either value or significance. It will be suggested later, however, that certain general principles do govern the clinical manifestations of this disease.

It is not now proposed to dwell in detail upon each of the many points for discussion arising out of these cases, and the particular aspect of the disease to which some consideration will be devoted in this paper is that introduced by the appearance of the motor symptoms mentioned in the heading to this chapter.

However, before any attempt is made to draw conclusions as to

their origin and nature in the particular cases recorded here, it may be of interest to turn to the numerous published descriptions of similar cases for any useful indications they may afford in this respect. The result is somewhat disappointing, for although many comprehensive accounts are to be found, particularly in the French literature, the clinical details given are so frequently coloured by the effort to identify widely differing muscular manifestations with the "secousses myocloniques" of Sicard and Kudelski [17, 18], that their probable nature can often only be surmised.

A case reported by Roger and Aymés (16) will help to illustrate this. It is described as a "syndrôme hémimyoclonique alterne séquelle d'encéphalite épidémique."

A young adult of 22 had an acute illness with convulsions followed by the development of a right-sided hemiparesis (face, arm, leg), with inco-ordination of the right upper limb, clonic jerkings of the right arm and slightly of the right leg. The right tendon jerks were increased and the right foot showed an "intermittent" extensor response. There is said to have been spasm of the left side of the face.

It is highly probable that we are dealing here with an encephalitic lesion of the left cerebral motor cortex with Jacksonian attacks in the right limbs. Again, we find Souques [21] expressing the view that "entre l'encéphalite myoclonique et l'encéphalite choréique, il n'y a, en effet, que des différences de mesure et de rythme dans les mouvements involontaires." Achard [1] has expressed a similar view. While, finally, Comby [8] describes as myoclonic encephalitis an illness with onset of fever, malaise, torpor followed by a phase of "myoclonus" and restlessness. The involuntary movements are described as being incessant, irregular and disorderly. Here we find the convulsive movements of what are almost certainly a Jacksonian fit described as "myoclonus," the view expressed that there is no real distinction between myoclonic contractions and choreiform movements, and in Comby's paper we see the latter described under the same name as the former.

From all this it is clear that the terms "myoclonus" and "myoclonic" in connection with this disease, though precise enough in Sicard's hands, have in those of others ceased to have a specific meaning and frequently serve only to obscure the characters of the involuntary contractions and movements described in clinical reports of this disease.

In these circumstances it may be of value to consider the original

meaning of the term myoclonus. It was first employed by Friedreich in 1881 to describe certain features seen in the disease he named paramyoclonus multiplex. "This affection," states Oppenheim, "is characterized by clonic contractions affecting mainly the muscles of the extremities and trunk, and rarely if ever those of the face. The contractions are short and lightning-like; they involve a small number of muscles which do not have a synergic action. The effect of the contractions in moving the limb is therefore slight or entirely absent. The contractions involve the muscles of the two sides of the body almost equally; they may be symmetrical, but not synchronous or rhythmical. The various twitchings, of which there may be sixty to one hundred a minute, are separated by intervals of varying duration. They affect a single muscle, which cannot be voluntarily contracted by itself, e.g., supinator longus. They may even be limited to parts of a muscle . . . active movements, which as a rule are unaffected, have a tranquillizing, soothing effect upon the spasm. Emotion has the opposite effect. The twitching diminishes when the attention is distracted, and disappears entirely during sleep." It may be added in conclusion that no discoverable lesion of the nervous system underlies the phenomena thus named and described.

In his first paper on the subject [17], Sicard describes the contractions in his myoclonic encephalitis as "secousses musculaires, brèves, rapides, explosives, à type rythme électrique, qui siègent sur la musculature des membres, de la face et du diaphragme, tantôt myocloniques, frappant un muscle ou un groupe de muscles, et ne s'accompagnant pas de contractions fibrillaires. Les algies disséminées persistent."

A comparison of Oppenheim's account with that of Sicard reveals the fact that the muscular contractions in the two cases bear but a relative resemblance, show certain distinct points of difference, and as far as our present knowledge goes appear to differ completely in cause.

Even, therefore, if we allow its aptness, the term myoclonus has but a limited sphere of usefulness in the study of lethargic encephalitis, and unless it is employed in Sicard's sense alone, and as a purely descriptive term without pathological basis, it had almost better be abandoned.

Nevertheless, from the descriptions given by those observers who use the term precisely we may obtain some useful information as to the probable site of the origin of these clonic contractions. Perhaps the most direct indication is to be found in two cases reported by

Sicard [17, 19], in which clonic contractions in the upper limbs gave place after several days' persistence to a paresis of peripheral type, namely, extensor weakness with wrist-drop. In one of these it is recorded that supinator longus was spared. While Sicard did not observe fibrillation associated with these contractions, such a combination has been recorded by several observers (Carnot et Gardin [6]). Again, we may note the random distribution of the clonic contractions, their frequent association with neuralgic pains of peripheral and spinal root distribution (often coinciding with that of the muscular contractions in topography). All grades of rhythmic muscular excitation have been recorded; fibrillary twitching, fascicular twitching (paramyoclonus) and contractions of whole muscles and muscle groups. These features all point to the lower motor neurone as the seat of an irritative or exciting lesion. According to Netter, "myoclonic encephalitis" marks the appearance of irritative symptoms in a disease where previously paralytic symptoms have predominated. However, neurologists have become cautious of explaining involuntary movements, and in some instances even pains, by postulating irritative lesions. Indeed, we know that the pain of the thalamic syndrome and the involuntary movements of tremor and athetosis are probably not so caused, but depend upon the release of lower centres from control. They are a reaction, an abnormal over-activity of intact portions of the nervous system when higher controlling mechanisms are out of action from destruction or otherwise. In the present instance it is extremely unlikely that we are dealing with any such release mechanism, and for the reasons given above it seems more probable that in the case of these clonic fibrillary, fascicular and muscular contractions we see the expression of an irritation or excitation of the lower motor neurone. The occurrence of Jacksonian fits indicates that the virus of lethargic encephalitis can and does act in this way upon nerve cells.

In considering the seven cases recorded in this paper, it is not proposed to discuss all the motor symptoms described, such as the Jacksonian fits of Case 1, the tremor and rigidity of Case 2, or the cerebellar symptoms of Case 4. These are in no way peculiar to lethargic encephalitis, nor does their occurrence in this disease throw any fresh light on their nature or origin. It may be of interest, however, to discuss briefly the choreiform movements of Case 3 and the fibrillary and muscular twitchings seen in Cases 5, 6, and 7.

In the preceding paragraphs and in the commentaries on the three last named cases reasons have been given for regarding the so-called

myoclonic contractions and the fibrillary and fascicular twitchings with which they are frequently associated, as evidence of an irritative or exciting action of the virus of the disease upon the lower motor neurone; that is, upon the cells of the peripheral motor nerves. It is now further suggested that the extreme psychomotor excitement of certain cases of lethargic encephalitis is another example of this action of the virus, in this case upon the highest cerebral centres. If this view be correct we see in the psychical and in the choreiform motor manifestations, in the Jacksonian fits, and in the rhythmic contractions and fibrillary twitchings of various muscles and parts of muscles the expressions of irritation or abnormal excitation of neurones in each of Jackson's three physiological levels of the nervous system.

There are many reasons for regarding the choreiform agitation (the "folie musculaire" or the "syndrome choréique à grand fracas" in the expressive language of the French observers) of such a case as the third of the present series as being irritative rather than dependent upon a release mechanism. Its constant association with great psychical excitement, its frequent association with the clonic muscular and fibrillary contractions already discussed (Labbé et Hutinel [13], Sicard [20], Harvier et Levaditi [11], Ardin-Delteil et Raynaud [2], Kahn [12], Dupouy [9], and Bourges et Marcandier [4]), and its rapidly fatal termination in most instances in exhaustion, coma and death all point to its being of this nature. While the disorderly, varying and complex character of the movements not only indicates that they arise in centres of high physiological level, but also marks them off clearly from the more orderly involuntary movements of the tremor and athetosis varieties, which are accepted as depending upon removal of control over subordinate motor mechanisms.

Finally, if we can accept this view of the irritative origin of these motor phenomena another remarkable point claims attention. It is that the virus of lethargic encephalitis in its paralytic action on the nervous system has always shown a characteristic and marked selective affinity for certain groups of motor neurones in the basal ganglia and brain-stem, producing the familiar basal ganglia and midbrain types of the disease with their associated lethargy. It does not cause widespread random paralyses, such as we meet in acute poliomyelitis. In its exciting or irritating effect, on the other hand, this selective affinity is totally absent, and it seems that neurones of every level physiologically considered, or, speaking anatomically, every part of the nervous system from cerebral hemispheres to spinal roots, are indifferently attacked.

Thus the polymorphic clinical characters of lethargic encephalitis are in large measure due to this second action of the virus, and we have in effect irritative and paralytic forms or stages of the disease. Each possesses clinical features of its own. Both may be present in a given case, and they occur in numerous and widely varying combinations, or they may occur singly. However, even in purely paralytic or negative forms of the disease, the involvement of the nervous system is probably more widespread than the focal symptoms indicate. The frequent general muscular hypotonus and diminution of tendon jerks of the lethargic paralytic cases would suggest this view.

Nevertheless, the striking contrast between these two effects of the virus on the nervous system raises a question which it must remain for pathological investigation to answer; namely, whether we may not be dealing with a complex and varying virus containing more than a single active component.

IV.—Summary and Conclusions.

The protean manifestations and the remarkable clinical diversity of lethargic encephalitis are discussed, and it is suggested that the numerous schemes of classification and description adopted, though inevitable in the early and growing stages of knowledge of so polymorphic a disease, are in fact tending to confuse rather than to lend precision to our conceptions of this malady, for practically every case presents, either simultaneously or at some phase of its course, the features of several clinical "types." The example of "myoclonic encephalitis" is discussed as demonstrating the misleading results of a purely symptomatic basis of classification and description.

A natural grouping of the symptoms of any toxic infective disease of the nervous system is given, and it is suggested that description, and as far as possible classification, upon an anatomical basis—that is, using the focal nervous signs for the purpose of defining types—would obviate much of the confusion at present to be found in the literature, and would at least ensure a uniform terminology in descriptions of the disease.

Acute poliomyelitis and lethargic encephalitis are contrasted in the light of the scheme referred to.

The points thus discussed in the first chapter are illustrated by a small series of personally observed cases, and an account of various positive motor symptoms is given. It seems probable that the psychomotor excitement of the "choreiform" manifestations of lethargic

encephalitis, the Jacksonian fits, and the rhythmic fibrillary, fascicular and muscular contractions collectively grouped by various writers as "myoclonic" symptoms are in fact the expressions of an irritative or exciting action of the virus of the disease upon neurones of the three physiological levels (Hughlings Jackson) of the nervous system, and that such symptoms can be localized on the same principles as are employed to localize negative or paralytic symptoms. If we accept this view of the irritative origin of the motor symptoms under discussion, a striking feature emerges: namely, that where the virus produces negative or paralytic symptoms it has, ever since the first appearance of the disease, shown a definitive and characteristic selective action on the cells of the basal ganglia and on those of certain motor nerves in the brain-stem, producing the familiar basal ganglia and midbrain types of the disease with its associated lethargy. On the other hand, in its irritative or exciting effect the virus appears to act equally on any and every part of the nervous system, from cerebral hemispheres to spinal roots, hence the polymorphic character of cases showing what we may call irritative symptoms. It is such cases that present the many difficulties in any attempt to classify lethargic encephalitis into clinical types. Possibly we may be dealing with a complex virus in which more than one active component exists.

REFERENCES.

[1] ACHARD. *Bull. et Mém. Soc. méd. des Hôpitaux de Paris*, 1920, t. xxxvi, p. 712.
[2] ARDIN-DELTEIL et REYNAUD. *Ibid.*, 1920, t. xxxvi, p. 341.
[3] BATTEN, F. E. *Brain*, 1916, vol. xxxix, p. 175.
[4] BOURGES et MARCANDIER. *Bull. et Mém. Soc. méd. des Hôpitaux de Paris*, 1920, t. xxxvi, p. 685.
[5] BUZZARD and GREENFIELD. *Brain*, 1916, vol. xlii, p. 305.
[6] CARNOT et GARDIN. *Bull. et Mém. Soc. méd. des Hôpitaux de Paris*, 1920, t. xxxvi, p. 125.
[7] CLAUDE. *Ibid.*, 1920, t. xxxvi, p. 269.
[8] COMBY. *Ibid*,, 1920, t. xxxvi, p. 446.
[9] DUPOUY. *Ibid.*, 1920, t. xxxvi, p. 759.
[10] ELLIS, A. W. M. *Lancet*, 1920, vol. ii, p. 114.
[11] HARVIER et LEVADITI. *Bull. et Mém. Soc. méd. des Hôpitaux de Paris*, 1920, t. xxxvi, p. 310.
[12] KAHN. *Ibid.*, 1920, t. xxxvi, p. 542.
[13] LABBÉ et HUTINEL. *Ibid.*, 1920, t. xxxvi, p. 194.
[14] LEREBOULLET et FOUCART. *Ibid.*, 1920, t. xxxvi, p. 391.
[15] NETTER. *Ibid.*, 1920, t. xxxvi, p. 441.
[16] ROGER et AYMÉS. *Ibid.*, 1920, t. xxxvi, p. 244.
[17] SICARD et KUDELSKI. *Ibid.*, 1920, t. xxxvi, p. 94.
[18] *Idem.* *Ibid.*, 1920, t. xxxvi, p. 121.
[19] *Idem.* *Ibid.*, 1920, t. xxxvi, p. 123.
[20] SICARD. *Ibid.*, 1920, xxxvi, p. 260.
[21] SOUQUES. *Ibid.*, 1920, t. xxxvi, p. 552.

ON DECEREBRATE RIGIDITY IN MAN AND THE OCCURRENCE OF TONIC FITS.

BY S. A. KINNIER WILSON.

For a number of years certain analogies between the results of experimental mesencephalic transection in apes and other mammals and occasional clinical phenomena in man, in cases where cortical function is completely in abeyance, have appeared to me worthy of consideration. Such cases, usually of an acute nature, do not occur with any frequency, and since 1905 only some half dozen of this particular character have presented themselves, but their main features exhibit similarities so definite as at once to suggest a more or less identical origin and localization of the symptoms. Briefly, the type of case herein described and analysed is one in which there is evidence of withdrawal of cortical control in the form of unconsciousness or semi-consciousness, the result commonly of cerebral hæmorrhage—submeningeal or intraventricular, or both—or of meningeal inflammation, or from the effect of certain intracranial tumours; also, for that matter, of hysteria. Coupled with this impairment of consciousness has been the appearance of tonic rigidity of trunk and limbs, minutely resembling experimental decerebrate rigidity, to which frequent exacerbations in the form of tonic or postural fits, accentuating and exaggerating the background of tonic posture, are superadded.

It will be shown, further, that in certain types of organic and functional nervous disease these postural attitudes may be adopted involuntarily with full consciousness, and that partial or fragmentary types also occur. To this last statement much importance is attached, since the hypothesis to be subsequently developed is, that the involuntary postures of a limb or segment of a limb in many forms of nervous disease, as well as the transient positions assumed in involuntary movements such as chorea or athetosis, are in reality simply parts of the complete decerebrate attitude.

The material may be divided into two sections as follows :—

I.—(1) Cases of decerebrate rigidity or attitude and tonic fits combined.

(2) Cases of decerebrate attitude without tonic fits.

(3) Cases of tonic fits without persisting decerebrate attitude.

These three groups include only those cases where the phenomena appear in association with loss of consciousness; their significance is enhanced by the light they throw on the second section of the material:—

II.—(1) Cases of decerebrate attitude in conscious life.
(2) Incomplete, limited, or fragmentary cases.
(3) Cases of decerebrate attitude in the course of conscious involuntary movement, especially chorea and athetosis.

I wish here to express my indebtedness to various colleagues who have kindly allowed me to examine or to refer to cases which have been under their care, and to acknowledge at the outset the debt this paper owes to the genius of Dr. Hughlings Jackson and of Professor Sherrington. It has proved a matter of considerable difficulty to collate from the literature analogous cases to those herein described, mainly, no doubt, because of the usually quite incidental reference to the phenomena and the variety of the headings under which such cases might be expected to be found. Those of which a *précis* is given later provide valuable confirmation of the general views expressed in this article.

To appreciate the significance of the clinico-pathological cases more readily it is desirable to cite first the results of experimentation.

The salient features of experimental decerebrate rigidity, as contrasted with the state following on transection below or in the lower half of the medulla, are given by Sherrington [15] as follows, the latter condition being described first:—

"If in a monkey or cat transection below or in the lower half of the bulb has been performed, the animal . . . . hangs from the suspension points with deeply drooped neck, deeply drooped tail, and its pendent limbs flaccid and slightly flexed . . . . On giving the hand or foot a push forward and then releasing it the limb swings back into and somewhat beyond the position of its equilibrium under gravity; and it oscillates a few times backward and forward before finally settling down to its original position.

"To this condition of flaccid paralysis supervening upon transection in the lower half of the bulb the condition ensuing on removal of the cerebral hemispheres (mesencephalic transection) offers a great contrast. In the latter case, the animal, on being suspended just in the same manner as after the former operation, hangs with its fore-limbs thrust backward, with retraction at shoulder-joint, straightened elbow, and some flexion at wrist. The hand of the monkey is turned with its palmar face somewhat inward. The hind-limbs are similarly kept

straightened and thrust backward ; the hip is extended, the knee very stiffly extended, and the ankle somewhat extended. The tail, in spite of its own weight . . . is kept either straight and horizontal or often stiffly curved upward. There is a little opisthotonos of the lumbo-sacral vertebral region. The head is kept lifted up against gravity, and the chin is tilted upward under the retraction and backward rotation of the skull. . . . . When the limbs or tail are pushed from the pose they have assumed, considerable resistance to the movement is felt, and, unlike the condition after bulbar section, on being released they spring back at once to their former position and remain there for a time even more stiffly than before."

Attention is specially directed to the posture of the fore-limbs in extension-pronation, and to the head-retraction and opisthotonos.

We may now pass to an examination of cases observed in man.

I.—(1) Cases of Decerebrate Rigidity or Attitude and Tonic Fits combined.

Case 1.—W. S., aged 19, shopman, was admitted to the National Hospital on June 19, 1905, under the care of Dr. J. A. Ormerod.

For at least four weeks previous to admission he had suffered from severe headaches, mainly frontal. About 11 p.m. in the evening of June 11, 1905, he suddenly "felt his head bad" and fell in the street unconscious, remaining so for about twenty minutes; thereafter, he complained of splitting headache across the forehead. During the night he had a fit with convulsions, but details were not forthcoming; it was followed during the next twelve hours or so by about nine other fits, apparently of a general epileptiform nature. In the course of the week before admission he frequently vomited and complained of giddiness, and was twice incontinent of urine. On getting up and attempting to walk he was stated to have staggered badly.

On admission to hospital his general condition was as follows : He lay on his back in bed, frequently passing his right hand over his forehead in a half-unconscious fashion. All questions were answered rationally, but usually in monosyllables, without dysarthria. He was drowsy and indifferent to his surroundings : frequently he gave long-drawn sighing expirations : frequently, also, he would snort and spit. While he often moved the right limbs about, the left were immobile; the left arm was kept adducted and flexed at the elbow, and the left leg adducted slightly at hip and flexed slightly at knee.

Well-marked double optic neuritis was present. The pupils reacted quickly to light. There was apparently some general weakness of all ocular movements, but the patient's state did not admit of satisfactory testing. In voluntary movements the right side of the face always moved before the left. The tongue was protruded in the mid-line.

In addition to the apparent paralysis of the left limbs, it was found that the left arm showed distinct rigidity and resistance to passive movements, and the left leg similarly. No sensory changes were detected. The arm jerks were equally diminished on the two sides. The abdominal reflexes were much down on the left. Knee and Achilles jerks were diminished on both sides, and a well-marked double extensor response was readily obtained, but no ankle clonus could be elicited. Occasional incontinence of urine was noted.

By lumbar puncture 20 c.c. of fluid were drawn off under considerable tension, pale, reddish-yellow in colour, quite translucent, and obviously hæmorrhagic. There was no difference between the first drop and the last.

The next day the patient was able, after much encouragement, to move the left arm a little at shoulder and elbow, and to make a feeble grasp with the left hand. On the other hand, the facial asymmetry on volitional movement was accentuated. Ocular movements were still imperfect; the sixth nerves were apparently weaker than the others, slight internal strabismus being noted with the eyes at rest. The knee-jerks were further diminished, especially the left, and the double extensor response persisted.

A diagnosis of cerebral tumour was made, probably right frontal, and, further, it was presumed that for some reason slight hæmorrhage into and from the tumour had taken place when the symptoms had become acute just prior to admission.

At this point, however, they changed, and presented the following striking clinical picture.

On the same day, June 23, 1905, at 11.10 p.m., I was called to see the patient.

He was lying on his back, with head turned slightly to the right, and was quite unconscious. The arms were extended by the sides, slightly flexed at the wrists, and notably rigid. The legs were also extended and adducted, with toes pointed down and in. Respiration was extraordinarily laboured, the rate no more than five to the minute; inspiration was short, about three seconds' duration, while expiration was long drawn out, averaging nine seconds. It was noisy at first, gradually becoming fainter and fainter, then a sudden jerky inspiration completed the cycle. The pulse was 180, full, of moderate tension, and slightly irregular, sometimes thudding for several quick beats, then intermitting. With inspiration it always beat more quickly and strong.

The pupils were regular and equal, about 4 mm., and reacted to light. The corneal reflexes were absent. When the lids were lifted the eyes were seen to be fixed and staring straight in front. Skin and mucous membranes were flushed but not cyanotic.

11.15 p.m. Respiration remained about 6; pulse had fallen to 96, and was again regular. The knee-jerks could not be elicited on either side, but a smart patellar tap produced in each case a contralateral adductor jerk. The legs remained fully extended and rigid. The recti abdominis were also rigid Patient was incontinent of urine, and a minute later had an erection.

11.20 p.m. The eyes began to turn slightly downwards, and at the same time the right pupil dilated till it was almost twice the diameter of the left; it was now immobile to a bright light, though the left contracted as before. The plantar responses, up to this point absent, were now extensor on both sides.

11.30 p.m. The eyes gradually deviated into a definite and unmistakable skew-position, the right eye being down and in, the left eye up and out. Both pupils reacted again to light, though the right remained larger than the left. The skew-deviation was maintained only for a minute and a half, both eyes then slowly moving to a more symmetrical position, while the right pupil became of the same size as the other.

11.45 p.m. About this time both arms became rigid, the left rather more than the right. Both were adducted at the shoulder and strongly extended at the elbow, with pronounced internal rotation and hyperpronation; the wrists were flexed, so that the palms of the hands looked up and out, quite turned

FIG. 1 (Case 1).—The typical decerebrate attitude of extension-pronation.

away from the body (fig. 1). This striking position of the arms was henceforward maintained; at intervals the whole arm would stiffen still further, as it were, and the attitude become more accentuated, as if by waves of contraction passing down the musculature, but even when these waves relaxed the extensor-pronation position was not abandoned. The legs were as before, rigid, adducted, extended, feet inverted and great toes strongly dorsiflexed.

12.20 a.m. Another fit started. Patient suddenly became absolutely stiff in the decerebrate position, and his face reddened, while respiration became occasionally laboured, down to four per minute. It consisted of a short inspiration followed by a long-drawn-out expiration in the proportion of about 1 : 4. The pulse had become much more rapid again, 140 in rate, but it was fairly regular. There was no twitching of the limbs, but simply an intense tonic spasm accentuating the extensor attitude. The eyes were open, staring straight in front; pupils were dilated to their widest and did not react to light; corneal reflexes were absent on both sides.

FIG. 2 (Case 1).—The typical attitude at the summit of one of the actual tonic fits. The left forearm is less pronated than the right. Intense tonic contraction of the extensor musculature, with slight wrist flexion.

[For figs. 1 and 2 I am indebted to Dr. Colin Russel, of Montreal, who was in residence with me at Queen Square at the time.]

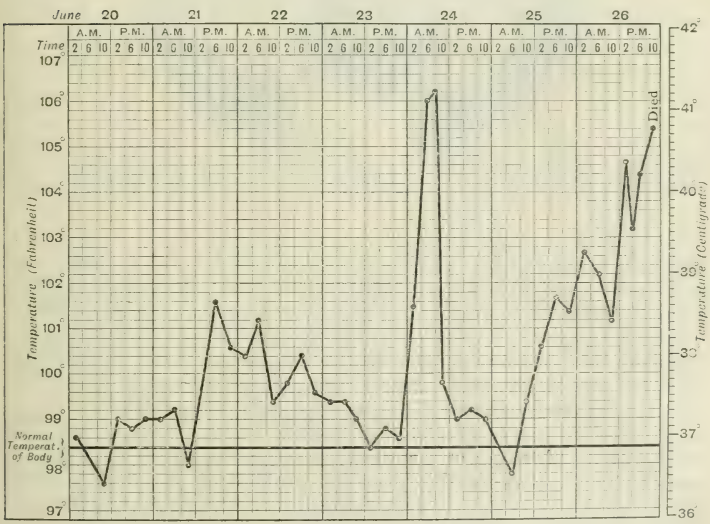

FIG. 3.

1.25 a.m. When the patient was seen again, after about an hour's interval, he was in the same extensor position, arms adducted at shoulders, extended at elbow, flexed at wrist, whole arm rotated inwards and hyperpronated; on the right the thumb was between the first and second fingers of the hand, but not on the left (fig. 2). The legs were rigidly extended, feet inverted, great toes dorsiflexed. Respiration 9, pulse 130.

For the remaining two days of the patient's life the phenomena continued at very frequent intervals without cessation. It appeared that any handling of the patient, e.g., during sponging (for with the decerebration high irregular temperature occurred, see fig. 3), precipitated the "attack," which consisted,

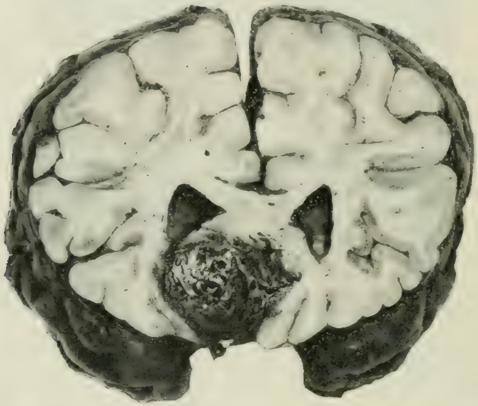

FIG. 4 (Case 1).—Position of vascular glioma (right frontal), whence hæmorrhage spread through the entire ventricular system.

approximately, of a period of Cheyne-Stokes' breathing for about one minute, associated with intense extensor rigidity of all limbs, inversion and hyperpronation of arms, flexion of wrists, thumbs between first and second fingers. The stiffening of the limbs preceded the onset of the altered breathing by a second or two; the face flushed slightly: the eyes always opened widely and the pupils dilated. In the feet there was usually flexion of the small toes, while the great toes were dorsiflexed proximally and flexed distally.

Death ensued after almost exactly seventy-two hours of complete decerebrate rigidity, with many scores of tonic fits. Post-mortem, a vascular glioma was found on the under-surface of the right frontal lobe (fig. 4), from which hæmorrhage had taken place, making its way right through the ventricular system and under the basal meninges, and spreading beyond the cranial cavity to the spinal theca. The third and fourth ventricles were blocked with blood.

The phenomena exhibited by the patient during these hours resemble in minute particulars those of mesencephalic transection in experimental animals. Until the hæmorrhage and increasing pressure killed him he was little or no more than a decerebrate preparation.

Case 2.—L. C., aged 34, a soldier, was admitted to hospital at 10 p.m. on the evening of January 29, 1918, with the history of having had a "stroke" earlier in the evening.

On admission he was conscious, and was able to speak fairly clearly. The left face, arm, and leg were not moved voluntarily. Complaint was made of severe pain in the head, which he frequently rubbed with his right hand. The pupils were of medium size, equal, and reacted to light. The mouth was drawn slightly to the right. All the deep reflexes were brisk, the knee-jerks excessively so, and there was double ankle clonus, with a double extensor-plantar response.

About 1 a.m. the patient suddenly became quite unconscious, and shortly after a series of convulsive attacks commenced and continued uninterruptedly for many hours, till within a short time of the patient's death the same day, January 30, at 2.25 p.m.

These attacks were of the following nature :—

The whole of the musculature of trunk, arms and legs became rigid ; the arms and legs were extended to the fullest extent, the arms by the sides and the legs together. In addition, both arms were strongly inverted and hyper-pronated, while the hands were clenched and the wrists slightly flexed. The legs were slightly inverted, the feet more notably so, with the toes pointing down and in. It was remarkable that notwithstanding the pre-existing paralysis on the left side, the musculature of both sides was equally and symmetrically involved in the tonic fits. While the musculature was in this rigid condition, fresh "waves," as it were, of tonic contraction passed through it, accentuating for the moment, if that were possible, the already existing posture.

The duration of each attack was less than half a minute. All through the night and far on into the next day these tonic fits continued, the patient never regaining consciousness. The interval between each was simply a few minutes.

At the autopsy recent fluid blood was found exuding from the pial roof of the fourth ventricle and spreading over the corpus callosum and over the whole of the basal surface of the hemispheres. On section an extensive recent hæmorrhage was seen to have ploughed up the interior of the right cerebral hemisphere and to have burst into the lateral ventricle, and so to the opposite lateral ventricle and right down the chain of ventricles to the fourth and through the foramina in its pial roof. The blood had also passed down the surface of the cord. There was obvious syphilitic disease of the cerebral vessels, and a small unruptured aneurism was found on the right middle cerebral artery. The free ruptured ends of the lenticulo-striate set of vessels on the right side lay in blood clot, but no aneurisms were found on them.

Case 3.—L. F., aged 39, married woman, was admitted to the National Hospital, under the care of Dr. James Taylor, on January 27, 1905.

In July, 1904, the patient began to suffer from severe headache and pain in the left occipito-parietal region, with giddiness.

In January, 1905, some weeks before admission, she noticed that her articulation was becoming defective, and within a few days the right arm and leg became distinctly weak, and the seat of occasional paræsthesiæ.

On her admission, moderate optic neuritis of both discs was found. There was well-marked weakness of the right side of the face, for both voluntary and involuntary movements, though less for the latter than the former. The patient lay in bed with the face turned to the left, the chin slightly up, the occiput to the right and rather down towards the right shoulder. The right arm was closely adducted to the chest, elbow at a right angle, forearm incompletely pronated, wrist extended, fingers and thumb slightly flexed. The whole limb was in a state of extreme hypertonus, and only slow and very weak voluntary movements were obtained in the muscle groups. The right leg was fully extended at hip, knee, and ankle, strongly hypertonic, and immobile.

A very brisk jaw-jerk was obtained, and though the patient did not complain in any way of the left limbs, and their volitional power seemed unimpaired, yet both abdominal reflexes were absent and the plantar response was definitely extensor on both sides.

On the fourth day after her admission a series of clinical phenomena of an unusual nature commenced and continued till her death five days later.

It was observed on the forenoon of January 31 that the patient was becoming drowsy and at one o'clock the left arm and leg suddenly passed into a rigid tonic spasm and relaxed again after five minutes. A similar tonic spasm of the left side occurred at two o'clock, of about the same duration. At six o'clock she was absolutely unconscious, the pupils widely dilated and immobile to bright light, respirations so shallow and noiseless as to be almost imperceptible, rate 16; pulse only 42, and slightly irregular. The patient lay immobile on her back, with limbs fully extended, and unexpectedly limp, only the slightest resistance to passive movement being noted in the joints. Within a brief period, however, respiration suddenly became noisy, irregular, and cogwheel or sobbing in character, and at the same time the extended limbs passed into a state of extreme rigidity in the decerebrate attitude; the upper extremities were adducted and internally rotated, the forearms strongly hyperpronated, so that the backs of the hands faced each other; trunk and neck were extremely rigid. and the latter slightly retracted; the legs were similarly in full extension, heels drawn up, toes pointing down and feet inverted. From time to time during the next few hours the rigidity waxed and waned, within moderate limits, but by 1.30 a.m. it ceased and the patient's limbs became once more relaxed, though the attitude of extension and pronation was unaltered.

During the next day the same alternation of tonic fits of all the muscles of the body with relaxation of tone occurred at intervals. That morning, when

the mouth was widely opened with a gag, it was seen that the uvula and soft palate were the seat of a rhythmical contraction and relaxation at the rate of about 120 a minute, entirely symmetrical on the two sides, there being no deviation of the raphe. This so-called "palatal nystagmus" continued without break or intermission for hours, and was re-observed at intervals all day. It is worthy of special attention.

On February 4 the limbs were flaccid and the patient apparently lifeless, respiration being imperceptible. Yet the extensor attitude never changed.

Death ensued the same day. At the post-mortem examination a tumour of the mesencephalon was discovered, invading the upper part of the pons. It proved to be a glioma.

Case 4.—M. B., a little girl, aged 6 years, was admitted to the National Hospital under my care on March 13, 1920.[1]

The history as given by the child's mother was that some weeks previously the patient had caught a cold, become rather deaf, and developed earache, which was followed a week later by headache, vomiting, and a rise of temperature. After three days of high fever her condition rather improved, but the headache and the vomiting persisted; actual otorrhœa never occurred. During the week previous to admission the symptoms became aggravated, and mild delirium set in, with restlessness and talkativeness. A squint also developed. The child was practically unconscious on admission to the hospital.

Neurological examination revealed hyperæmia and some blurring of the discs, and bulging of the tympanum of the right ear. Almost complete right and incomplete left ophthalmoplegia, interna and externa, with weakness of the left side of the face, was noted.

The child lay in a semi-conscious state with eyes half closed, mouth open, and head turned to the left. The left arm was flexed acutely at the elbow, adducted at the shoulder, and extended at the wrist, the fingers being flexed; the right arm lay in an easy extended position; the legs were fully extended and adducted, and the toes pointed downwards (fig. 5). From time to time the child moved her limbs in a vague and restless way, moaning the while, and occasionally opening her eyes.

At intervals during the twenty-four hours of her life which remained she exhibited tonic fits in a highly characteristic form. These were most commonly bilateral, but occasionally more severe on the left side than on the right.

A brief period of excitement and moaning ushered in the attacks, which consisted of sudden powerful opisthotonos and head retraction, the neck straightening and the occiput nearly touching the shoulders. The left arm relinquished its flexed attitude and became strongly extended, the forearm over-pronated, the wrist extended to a complete right angle, the first and second fingers extended and the third and fourth flexed, while the thumb was flexed

[1] For the notes of this case I am indebted to my house physician, Dr. M. A. Blandy.

FIG. 5 (Case 4).—Incomplete decerebrate attitude in the intervals between the tonic fits. On the occurrence of the latter the left arm passed to full extension-pronation while the head straightened and was mesially retracted.

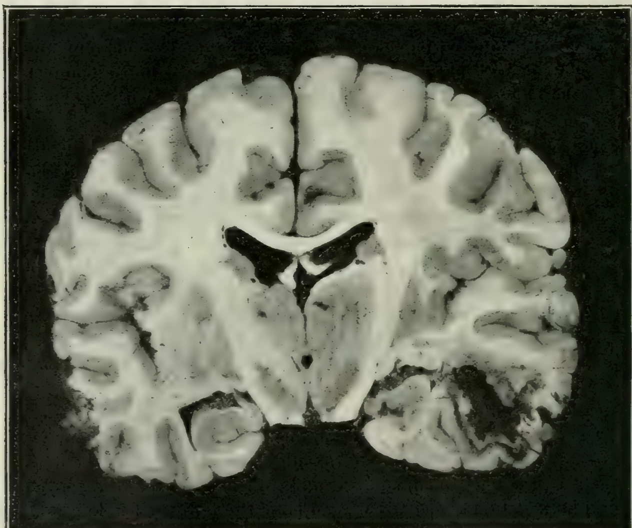

FIG. 6 (Case 4).—Shows position of temporal abscess from which the ventricular system became infected.

into the palm. The legs were in fullest extension with heels up and toes down, and the right arm was as the left, except that it was rather less inverted and the hand was in the form of a fist. Trunk and limbs alike were absolutely rigid. During the attacks, which lasted about ten to fifteen seconds, respiration quickened, and was accompanied by slight expiratory groaning. No clonic movements occurred at any time in the course of the attack, cessation of which was followed by relaxation of the hypertonicity and resumption of the original attitude described above.

Examination of the reflexes showed absence of the abdominals, activity of all the deep reflexes, bilateral extensor plantars, and a crossed extensor from the right to the left.

Lumbar puncture revealed a somewhat turbulent cerebrospinal fluid, in which organisms of the streptopneumococcus class were found. Death ensued within thirty-six hours of the patient's admission.

At the post-mortem examination there was general meningitis over the cortex and more marked along the base. The right middle ear was full of pus. Section revealed an abscess in the right temporosphenoidal lobe, whence, apparently, the ventricular system had become infected (fig. 6). There was but slight internal hydrocephalus.

Case 5.—M. C., aged $2\frac{3}{4}$ years, was admitted to the National Hospital under Dr. Ormerod on February 28, 1906.

Three weeks before admission the little girl had had a generalized epileptic attack, followed by another an hour or so later. Since, she had been apathetic yet irritable, occasionally giving vent to a cry. She had also become inattentive to her excretory functions.

On admission, the patient was drowsy but irritable, lying more or less constantly on her side with complete flexion of all her limbs, and with her back bent, so that she was literally "huddled up" in bed. The limbs could be passively extended at all joints, but the procedure brought out a certain amount of resistance in the flexors from hypertonus. The knee-jerks were active and the plantars flexor.

March 3, 1905: Patient comatose and roused with great difficulty. Occasional short spasmodic flexion movements of all limbs. Temperature 100·8° F.

10.30 p.m.: The arms remained flexed at all joints, but the legs had become fully extended at hip, knee, and ankle. Abdominal reflexes absent, knee-jerks active, double extensor response.

March 4, 1906: The extensor position of the legs was not maintained; they were sometimes flexed, sometimes extended.

March 6, 1906: The legs were now extended again, and so were the arms. The patient lay unconscious, with the arms extended by the sides and the forearms notably hyperpronated. The hands were clenched. The legs were also fully extended, with feet inverted and toes pointing down and in.

At frequent intervals tonic spasms passed down the musculature, re-

inforcing and accentuating the persisting extensor attitude of arms and legs. It was noteworthy that in these brief attacks the pronation of the forearms was always markedly increased. The trunk was extended, but not to an opisthotonic degree, nor was there more than slight retraction of the head.

This extensor rigidity, with occasional increase from transient extensor and pronation spasms, was maintained unchanged for the next few days, in fact up to the patient's death on March 11, 1906 (fig. 7).

Post-mortem: Typical tuberculous meningitis of the base, with moderate internal hydrocephalus.

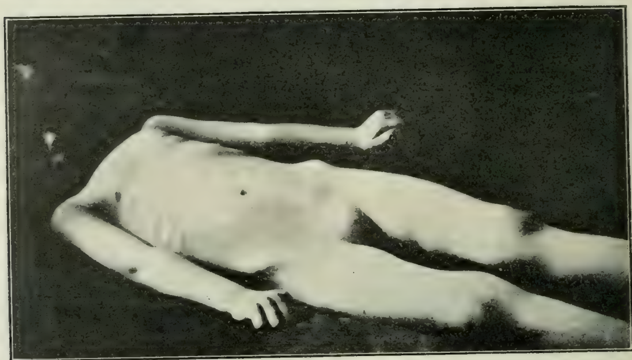

FIG. 7 (Case 5).—Decerebrate attitude maintained for several days *sub finem.*

A consideration of these five cases will suffice to illustrate the cardinal features of decerebrate rigidity in man, in its complete form. In each the decerebrate posture is reproduced with the exactness of a physiological experiment, and once it is appreciated it can always be recognized for what it is. The details of the attitude in man being on a larger scale, can be studied with greater ease than in mammals. One of its most significant components is the extension-pronation of the arms and the flexion of the wrists. We shall see later how it is just these components which are most likely to make their appearance in incomplete cases. The reader will also note that in all five examples the onset of tonic fits merely accentuated the already existing posture, showing that tonic fits are in reality attacks of decerebration, if the expression be allowed. The absence of any clonic element is of much significance. It may be taken to indicate that tonic and clonic movements have different origins and are the expression of the activity

of different motor mechanisms; one is characterized by phasic and the other by static activity.

From the pathological viewpoint it will already be apparent that in each instance the morbid process was so situated as to dissociate the cortex functionally from lower centres, which exhibit an activity of their own until such time as they themselves are overwhelmed.

It is particularly noteworthy that in many of the cases striking respiratory disorder ushered in the tonic attacks or persisted with the maintenance of the decerebrate posture.

(2) Cases of Decerebrate Attitude without Tonic Fits.

Case 6.—I owe this case to the kindness of my colleague at King's College Hospital, Dr. G. F. Still.

D. S., aged 15 months, was admitted to hospital, under the care of Dr. Still on February 28, 1920, with a history of "being ill" for six weeks. The chief clinical symptom was persistent vomiting after every meal for the previous three weeks. It was noted by the mother on the day of admission that the child's eyes seemed "fixed" and that its limbs were sometimes trembling and sometimes stiff. No fit had been observed, but for a day or more the infant was thought not to have recognized its mother.

On admission the optic discs were normal, the pupils reacted rather sluggishly to light, the fontanelle was flat. The child lay in an apparently unconscious condition with arms and legs extended and rigid, but without head-retraction. Kernig's sign was not obtained. From this date to the child's death, six days later, the attitude of decerebrate rigidity was maintained and indeed intensified. Thus the arms were fully extended, adducted, strongly hyperpronated; the backs of the hands faced each other, and at the same time the wrist and finger flexors were conspicuously contracted; the neck was stiff from muscular rigidity but was not retracted in any degree; the lower extremities were in full extension and adduction, with heels drawn up, toes pointing down, and feet slightly inverted (fig. 8). The abdominal reflexes could not be elicited, the knee-jerks were active and the plantars extensor. With a pulse of 150 was coupled a slow, sighing respiration. The child died quietly on March 9, after unconsciousness and decerebrate rigidity of some nine days duration.

Permission for a post-mortem examination was refused.

(3) Cases of Tonic Fits without Persisting Decerebrate Rigidity.

Less infrequent, possibly, than cases belonging to either of the two previous categories are those in which the occurrence of tonic fits is not associated with any maintenance of a decerebrate-rigidity attitude apart from what obtains during the fit itself. "Cerebellar fits," so-called—not

the best name to give them—have long been known, and since the days of Hughlings Jackson little has been added to his descriptions and interpretations. It is, however, desirable, not merely to bring the phenomena of tonic fits into line with what experimental physiology

FIG. 8 (Case 6).—Striking decerebrate posture of limbs and trunk, but with no head retraction. Photo taken from above. with child lying on back.

has taught us of decerebrate rigidity, but also to show that *it is possible to resolve these complete seizures into component elements, that partial, incomplete, even segmental tonic attacks may be met with, and that some at least of the more or less fixed attitudes of head and neck, trunk, and of one or other limb or part of a limb, encountered in certain basal, subtentorial and other lesions, are in reality functional fragments*, so to say,

of a non-cortical motor mechanism which finds its full expression in decerebrate rigidity and in complete tonic fits.

For the moment it is immaterial to our purpose whether these "tetanus-like seizures," as Jackson called them, are of organic or hysterical origin; a number of instances of each have come under observation.

Much the most striking of all is the following case, difficult though it be of exact diagnosis.

Case 7.—V. S., aged 18, was admitted to the National Hospital under the care of the late Dr. C. E. Beevor, on April 10, 1906.

Some months previously he had fallen down a flight of stairs on to his head, and though momentarily stunned and thereafter dazed for some hours he had not, apparently, really lost consciousness. Two weeks later he was thrown down some stairs, apparently in a "rag," struck his head again, and lost consciousness, but it is not known for how long. Thereafter he complained of pains in the head, shakiness, and drowsiness. Two months later he had an attack of influenza, and two days thereafter his first fit. These were of a peculiar character, described in detail below, and recurred on an average three or four times a week. They presented certain diagnostic difficulties, it is true, and opinion was divided as to their organic or hysterical nature, but it is important to note that the patient had had the attacks when in company and when alone in bed, indifferently, and that during his stay in hospital the efforts made to inhibit or cut short the attacks by the usual methods adopted in cases of hysteria were on each occasion a conspicuous failure.

On objective examination no signs of any organic disease of the central nervous system were found. The optic discs were clear and the reflexes in all respects normal.

The following is an account of a series of fits observed by myself.

When I reached the patient he was already lying in the semi-conscious condition succeeding a fit. He was breathing heavily, foaming slightly at the mouth, and was on his back. A moment later, he passed into a fit.

His arms went out in front of him, extended and hyperpronated, with hands clenched and wrists flexed. The eyes rolled up and the eyelids closed. The face appeared drawn, as if in a tonic spasm, while the arms and to a less extent the legs were absolutely stiff. Then the arms went out from the sides, and he began to rotate from left to right, so that he turned right round and lay on his face. This turning movement was slow and deliberate, and it was accompanied by slow stertorous respiration. At the same time as he turned his body became slightly curved anteroposteriorly, with the concavity forwards. He did not turn any further, but lay thus on his face. There was no clonic movement of any sort. Then in a few seconds came a relaxation, whereby the tonic spasm died down, and the limbs became completely atonic, flabby, so that they could be shaken about like flails. His respiration became more easy, and his colour less congested.

Nurse and I turned him round on to his back again.

A moment later, he passed into a second fit.

His corneal reflexes, tested at the commencement of this, were absent. The eyes deviated strongly upward, and at the same time the left arm went out into a tonic spasm, being away from his side and hyperpronated. At the same moment as his arm went out, he arched his body backwards, so that the occiput was down on the nape of the neck, and the muscles of the back in a powerful tonic contraction. The right arm was by the side, and it then contracted strongly in extension and internal rotation. With this, he again began to rotate, the left arm leading the way, and the trunk coming round after it from left to right, until he had come round on to his back again, nurse helping him, otherwise he would have been off the bed.

The same sequelæ, relaxation of the contractions and complete atonia, were observed. In the interim, the corneal reflexes returned, but he made no response to stimuli.

He then passed into a third fit.

This began by his rising off the bed completely, owing to strong contraction of the recti abdominis, &c., and in this anteroposterior plane he curved right forwards. Then his arms went away from the sides, the left before the right, both being powerfully rotated internally, and the hands clenched. The fists were strongly flexed on the forearms. The eyes were closed and the face drawn and quivering slightly. The under lip was curved, the corners of the mouth being depressed.

The position just mentioned was maintained for a moment, then he began to rotate slowly to the right, and at the same time to arch back into an opisthotonic position. A moment later, therefore, he was on his left side, his arms as before, his back most powerfully contracted, the head right back, the face looking upwards. Round he came still further, till he was on his face, and as he was breathing heavily we turned him on to his back again. There was no clonic movement whatever. I tried his jerks rapidly, and found they were all increased, the abdominals were diminished, especially on the left side, and there was a double flexor response. There was no incontinence and no biting of the tongue.

He sank a few seconds into the same atonic condition, with increased jerks, and lay motionless, salivating slightly.

Almost as soon as he had come round on his back the muscles relaxed and the limbs again became peculiarly atonic. He breathed deeply and his colour improved. There was no extensor response, but both abdominal reflexes were diminished.

A minute or two later, he put out both arms by his sides, separating his fingers, and then opened his eyes widely. His respiration quickened and he smacked his lips a little.

In this remarkable series of absolutely pure tonic fits, whatever their pathogenic basis, it is plain that the postures adopted were for the

major part precisely those of decerebration, especially in regard to the extensor rigidity with pronation of forearms and flexion of wrists, and the head retraction and opisthotonos. The changing nature of the movements, notably the occasional trunk flexion and the rotation, indicates that in persisting decerebrate rigidity the posture may be in part compounded of mutually antagonistic couples, which may be dissociated in the course of an actual tonic fit, and in part the result of one of these being for some reason overborne by the other. Reference is made to this consideration in a subsequent section. Where there is no persisting *structural* cortico-mesencephalic separation there is less likely to be fixity of posture.

Of several other cases of tonic fits associated with unmistakable organic disease the following may be selected as sufficiently illustrative.

Case 8.—The patient was a woman of 28 with a comparatively short history of headache, giddiness, vomiting, and optic neuritis. These general symptoms of intracranial tumour were not, however, accompanied by any definite localizing sign, although the persistent pain over the left side of the head warranted a left occipito-parietal decompression. During the five days between the operation and the patient's death she had several fits of a strictly tonic nature.

She suddenly became unconscious while the head wound was being dressed, both legs going out into strong extensor tonic spasm, the knees adducted and the feet powerfully plantar-flexed and inverted: the arms were similarly extended and adducted, with forearms hyperpronated on one occasion, but on another definitely rotated outwards, i.e., in a position of supination; the fingers were strongly adducted and extended, while the thumbs were flexed at the metacarpo-phalangeal joints and adducted across the palms. The head was markedly retracted and there was a distinct degree of opisthotonos of the spinal column. During the attacks, which lasted not more than 30 seconds, respiration always became very irregular. Immediately after the cessation of the tonic stage the limbs relaxed, seemed empty of tone, so to say, and could be moved about like flails.

At the post-mortem examination a glioma, of the size of a walnut, was found invading the cortex and white matter of the middle lobe of the cerebellum towards its left side, and of part of the left lateral lobe. From the operation wound and subcortical exploration then made blood had oozed into the left lateral ventricle and passed downwards.

It is obvious that the presence of the tumour alone will not explain the generalized bilateral tonic fits, and I attach more pathogenic importance to the results of the operative interference and the intra-ventricular hæmorrhage.

The occurrence of tonic fits as a hysterical manifestation is a neuro-

logical platitude, but their real significance for the purposes of this paper consists less in their psychogenic origin than in the morphological aspect they may assume. If we imagine the most superficial degree of hysterical dissociation to involve the transcortical neuronic level the movements released will be purposeful and complex, resembling normal motor activity except for full conscious participation therein; if, similarly, we assume functional disintegration to descend to lower neuronic levels we should expect to observe phenomena morphologically analogous to those of decerebrate rigidity, which is, as a fact, precisely the case. In this way the actual movements of certain moderately severe hysterical seizures receive a physiological explanation and illustrate the disorders of function which we have associated with organic decerebrate human preparations.

It seems unnecessary to cite any case in support of this contention, so commonly are the opisthotonic and extension-pronation postures found in hysteria. The reader is referred to one of Richer's drawings,[1] from which it will readily be seen that the attitude of the *arc de cercle* is strictly comparable to that following mesencephalic transection.

Scattered through the literature are a fair number of cases of what should be called "decerebrate rigidity in man," some of which have been observed with sufficient minuteness to be of documentary value. Of older cases, that recorded by Bastian [1], under the caption: "A Case of Apoplexy in a Boy, aged 15; Intraventricular Hæmorrhage, Convulsions, and Death in Four Hours," may be mentioned, and there are, of course, the original cases of Hughlings Jackson [11], some of which were republished in *Brain* (1906), and several to which he himself referred (Dreschfeld, Drummond, Bacon). One of the cerebellar cases of Stewart and Holmes [16] (No. 9 in their paper) is of particular value in this connection, and the recent article by Douglas Firth [6] may also be referred to. A good example of tonic fits in association with a large cyst of the cerebellum was reported by Eskridge [5]. Of particular interest are two papers by Bruce, one entitled: "A Case of Intracranial Aneurism in a Young Subject" [3], and the other, "Aneurism of the Anterior Cerebral Artery with unusual Prolongation of Life after Rupture; Autopsy" [4], and a third, published by Dr. Violet Turner [19], under the heading, "A Case of Prolonged Hyperpyrexia in a Child with a Mid-brain Tumour," of which three cases a précis follows.

[1] Paul Richer: "Etudes cliniques sur l'hystéro-épilepsie." Paris, 1881. Plate III, fig 2.

(1) *Bruce* (1904).

The patient was a miner, aged 27, whose chief complaint was of absolutely unbearable headache, but apart from slight congestion of the optic discs no objective neurological sign was discovered on minute examination. Ten days later he developed paresis of the right third and sixth cranial nerves. After temporary improvement, a fortnight from his admission the symptoms suddenly became acute again, and at 8.20 one evening he had a general epileptic fit, from which he never recovered consciousness. A second convulsion occurred at 9.15, and a third at 9.50. During the night numerous attacks of spasm occurred. For some hours these occurred about every five minutes, each period of spasm lasting about forty or fifty seconds. The spasm began with a gurgling sound in the throat; the breathing became accelerated (to 52 per minute) and noisy; and the pulse to 120 or 130. *The limbs became rigid: the arms were extended in front of the body and rotated so that the hands were placed back to back. The fingers were closed.*

The intercostal muscles and the diaphragm acted violently. In the intervals between the spasms the breathing was comparatively quiet and tranquil, and the limbs were placid. The pupils varied in size and reacted sluggishly to light. The patient died at 7 a.m.

At the post-mortem examination the base of the brain was covered by extensive diffuse subarachnoid hæmorrhage, spreading from the frontal region, optic chiasma, anterior surface of the pons and medulla, to the cerebellum. On opening the brain there was found a considerable quantity of recent coagulum in both lateral ventricles, but more abundant in the left. On separating the frontal lobes there was found, arising from the anterior communicating artery, a spherical aneurism about the size of a small hazel nut. It had burst on the left side and there was considerable softening and infiltration of the tissues of the left frontal lobe. The blood had passed both upwards into the ventricular system and into the subarachnoid space, where it had become widely diffused.

(2) *Bruce* (1908).

The patient was a smith, aged 33, who was admitted to hospital in a state of unconsciousness, which was said to have lasted about two hours.

On admission, he lay with arms and legs extended; the eyelids were half closed, the pupils contracted, circular, equal in size, and reacted to light only to a slight degree, if at all. Breathing was irregular both in rhythm and in depth of inspiration. The arms lay along the sides of the trunk, but *there were frequent spasms in which they were forcibly extended at the elbow and drawn to the side of the chest, or even in front of it. The forearms were very forcibly pronated, so that the backs of the hands were directed towards each other. The wrists were flexed, and the fingers were fully flexed, causing the hand to be tightly clenched, the thumbs being strongly adducted against the index fingers. During these tonic spasms the lower extremities were tonically extended, and*

there was extension at the ankle joints and inversion of the feet. The spasms lasted a few seconds, and were usually accompanied by a loud expiratory effort and puffing out of the cheeks, but the facial muscles were otherwise not involved. The head tended to rotate to the left side, and the chin was approximated to the left shoulder.

There was ankle clonus on both sides, but both plantar reflexes were flexor in type.

During the day the spasms continued. By 3.30 *the respirations were more definitely of a Cheyne-Stokes character, and at the height of the cycle the arms and legs became rigid and the back tended to be arched.* Lumbar puncture was performed, and 25 c.c. of a brownish red fluid were drawn off under high pressure. This fluid was obviously composed of blood and cerebro-spinal fluid mixed. By 8 p.m. the spasms had ceased.

The patient lingered in a comatose state for eleven more days, when he died.

At the post-mortem examination a small aneurism, the size of a pea, was discovered on the left anterior cerebral artery. It had given way, and from it the blood had spread directly into the subarachnoid space, passing along in all directions, filling up the sulci and the subarachnoid cisterns, to some extent "seeking an entrance" into the fourth ventricle from the foramen of Majendie, and also passing along the spinal cord under the arachnoid. The blood had also ploughed right through the corpus callosum and had entered the lateral ventricles and had coagulated there.

(3) *Turner* (1916).

The case was that of a female infant, aged 18 months, who was admitted to hospital in an unconscious state. Two months before admission the child had stopped talking and become dull and listless. For two weeks the legs had been stiff and crossed, and the pupils dilated. For the last three days the stiffness had increased, and opisthotonos had been noticed one day before admission.

On examination the child was quite unconscious, with half-open fixed eyes. The whole body was rigid, and the back arched with some head retraction. All four limbs were rigidly extended, and there was adductor spasm of the legs. All the deep reflexes were exaggerated ; the plantar reflexes were extensor and the abdominal absent. After lumbar puncture and the removal of $2\frac{1}{2}$ oz. of clear colourless fluid under great pressure the rigidity diminished gradually, and for one day the limbs were almost flaccid. The following day the spasticity returned and the fontanelle became very tense. From this time onwards until death, no less than twenty-eight days later, *the child lay in a state of extreme rigidity in the extended position, the feet in a position of marked plantar flexion, and the thighs adducted. The upper limbs were so rigid that at times they did not rest on the bed ; the forearms were persistently pronated so that the palms were turned outwards, and the fingers flexed over the thumb,*

which was held across the palm. Actual opisthotonos was, however, only once present. The jaws were usually tightly clenched.

Mention may be made in passing of the extraordinary pyrexia in the case, reaching on one occasion 111° F. in the rectum, and 109° F. in the axilla.

Post-mortem, a solid rounded tumour was found situated in the mid-line in the region of the corpora quadrigemina (fig. 9). It did not involve the pons, cerebellum or optic thalami, but pressed forward into the aqueduct of Sylvius, causing a block, with resulting distension of the third and lateral ventricles. The tumour was apparently of a glio-sarcomatous type.

FIG. 9 (Turner's case).—The position of the tumour is such as to produce complete physiological decerebration at the level of the corpora quadrigemina.

These three cases in particular furnish ample corroboration of the connection between the peculiar symptomatology of decerebrate rigidity with postural fits and the neuronic level of the underlying lesions, and the identity of the postures in all confirms the support given by clinical observation to the findings of the physiological laboratory. The child in Turner's case was to all intents and purposes an experimental decerebrate preparation, and its survival for four weeks renders the case unique, as far as I have been able to discover, from this point of view.

It will doubtless occur to the reader that in various organic nervous conditions the phenomena under consideration make their appearance

in greater or less detail. Thus, not a few cases of Little's disease conform strictly to the decerebrate type. Attention may also be directed to the cases published by Sutherland and Paterson [17] under the heading. "A Type of Cerebral Mal-development (Forebrain Aplasia)." In these infants imperfect development of the cerebrum with relative conservation of the neuraxis below the basal ganglia has led, as might be expected, to the appearance of the decerebrate attitude, as can be seen by a glance at the figures in the paper.

Further, the familiar head-retraction of various forms of basal meningitis, with or without actual opisthotonos, is certainly but a part of the complete decerebrate posture. Reference will be made to this point again.

II.—(1) Cases of Decerebrate Attitude in Conscious Life.

There is no reason to doubt that the involuntary adoption, when consciousness is not in any way impaired, of an attitude corresponding exactly to the whole or any part of the decerebrate posture is to be attributed to the action of the same mechanism. The symptoms, it will readily be understood, are most apt to appear in circumstances

Fig 10.—Sir Charles Bell's drawing of a case of surgical tetanus.

under which the superior control of the corticospinal system is structurally or functionally defective; but, on the other hand, the characteristic postures are scarcely so likely to be maintained indefinitely as in the unconscious cases. There is, as it were, a conflict between two motor mechanisms, each attempting to jockey the other out of possession of the final common path.

I do not remember ever to have seen the assumption of the decere-

brate posture in hysteria during consciousness, though a case of this description is figured by Dejerine in the second edition of his "Sémiologie" (fig. 228, p. 544). In cases of tetanus the tonic spasms, consciousness being unclouded, are often closely analogous to those we have been considering, as Hughlings Jackson emphasized—hence his expression "tetanus-like seizures" for tonic fits, already alluded to. Fig. 10 is a reproduction of Sir Charles Bell's drawing of a case of surgical tetanus, observed by him on the retreat with Sir John Moore to Corunna. The flexion of the arms, in contradistinction to the usual extension of the typical decerebrate position, has, of course, frequently been noted in "cerebellar fits," and its meaning is discussed below.

FIG. 11 (Case 9).—Decerebrate attitude of upper limbs in a case of diplegia.

Cases of more or less Complete Decerebrate Attitude.

Case 9.—E. W., aged 2, was admitted to the National Hospital under the care of Dr. Risien Russell on March 1, 1920.

The child was a nine months' infant, born naturally, and breast-fed.

Apparently no particular notice was taken of her condition till she reached the age of six months, when her inability to sit up was remarked on. Her mother, nevertheless, admitted on being questioned that the child "had never used the arms properly" and that the legs tended to be stiff at the knees. The child had never walked.

On examination no impairment of function of the cranial nerves was detected. Intelligence, as far as could be ascertained, seemed good enough; body and head were well-proportioned. With support the patient was occasionally able to take a step or two in a very imperfect way, getting on to her toes and lifting her feet high. There was considerable spasticity in the legs and some varying rigidity of the trunk. Much the most noteworthy feature of the case, however, was the attitude of the arms, one not persistently maintained, but adopted involuntarily and interrupted only by voluntary movements. When the infant was lifted up or supported in a more or less vertical position the limb attitude was especially manifest, and it was often maintained for many minutes at a time (fig. 11). The arms were adducted at the shoulders and sometimes rather advanced, fully extended at the elbows and powerfully inverted and hyperpronated, with wrists extended and hands closed in flexion, presenting an absolute identity with the attitude of decerebrate rigidity already described.

Case 10.—H.O., aged 9, had been born at full time in a normal confinement. She was always weak in her arms and legs, and had not walked till she was three, and then very imperfectly. The history given by the parents was that she was slowly getting worse and that for six weeks she had not been able to walk at all.

When she came under observation her intelligence was noted to be very poor, and examination was rendered unusually difficult owing to her inattention.

What struck the observer was the extreme degree of volitional imperfection as regards movement: the child seemed to be unable to move any limb volitionally at all, and to succeed in doing anything to order only, as it were, by accident. The musculature of the body and limbs was distinctly rigid on passive movement, and the hypertonus was more noticeable proximally than distally. The abdominal reflexes were absent and the plantars extensor.

At one time the patient seemed to lie in a position of more or less generalized flexion, and at another, of generalized extension. In the former of these the trunk was bent forwards and the chin almost reached the chest; the arms were adducted, elbows flexed, forearms pronated, crossing each other on the chest; the thighs were flexed and adducted and the legs flexed at the knees. This huddled-up attitude seemed to be assumed mainly, though not solely, during sleep. When the patient was disturbed, or her limbs handled during an examination, she almost always passed into an extensor attitude (fig. 12), with head retracted, arms fully extended, inverted and hyperpronated to an

extreme degree: the legs were not, as a rule, fully extended at the hips or knees, though the feet were inverted, heels drawn up, and toes pointed down.

It should be mentioned that in addition to these attitudes the limbs were the seat of occasional slow spontaneous movements of an athetoid nature.

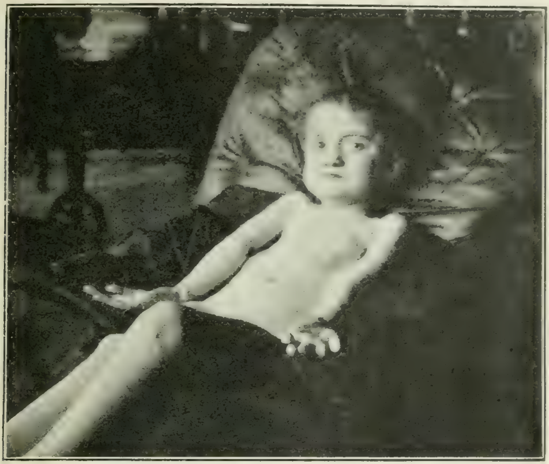

FIG. 12 (Case 10).—Extension-pronation attitude of upper limbs in a diplegic (see text).

(2) INCOMPLETE, LIMITED, OR FRAGMENTARY CASES.

Included in this section are cases where the decerebrate attitude is unilateral in arm and leg, or confined to one limb or segment of a limb. The more severe the degree of cortico-spinal interruption, corresponding to what obtains in actual decerebrate rigidity, the more likely is the unilateral attitude to resemble what is found in the complete cases, of which a number of instances have been furnished. It might be supposed, simply, that the ordinary hemiplegic attitude of arm and leg is a unilateral decerebrate posture, but this is the case only in a sense. One scarcely sees in experience the complete half attitude which would depend on the existence of complete unilateral cortico-spinal interruption, and this, owing to bilateral representation, cannot in fact occur. The upper extremity, again, assumes an attitude in flexion at the elbow, commonly, and not extension; nevertheless, the adduction at shoulder, pronation of forearm, and flexion of wrist and perhaps

fingers, are to be ascribed to the development, incomplete though it may be, of the decerebrate posture. It is important to recognize, in this connection, that in experimental unilateral decerebrate rigidity there are phenomena to be observed on *both* sides of the body. The following is Sherrington's [15] account of the condition:—

"If instead of both cerebral hemispheres one only, say the right, be ablated, the decerebrate rigidity appears, though not with the same certainty as after double ablation, chiefly on the same side as the hemisphere removed. The monkey, when slung after ablation of one—e.g., the right—hemisphere exhibits generally the following attitude. The right limbs are extended in the pose above described as characteristic for decerebrate rigidity, the tail is strongly incurvated toward the right, that is, its concavity is toward the right and its tip is also toward the right. It resists passive movement to the left, and if displaced thither immediately on being released flies back. The head also is pulled toward the right and retracted. The left forelimb . . . . is sometimes distinctly more flexed than would be expected in the paralysed condition of the animal; the left knee likewise. . . . . The contrast between the attitude of the crossed and homonymous sides is very striking . . . .

"Homonymous extensor rigidity consequent on ablation of one hemisphere is however neither so constant of production nor so persistent when it has appeared as the rigidity following bilateral ablation. After coming on it may totally subside and again reappear, and so on several times over. There would seem under these circumstances a struggle between two conflicting influences, as though a tonic influence from the still intact crossed hemisphere at times overcame and at times was overcome by another opposed influence from a lower centre. Some amount of extensor rigidity on the side opposite to the lesion is not uncommon."

Through the kindness of Dr. Poynton I am able to refer to a valuable case which he has afforded me every opportunity of examining, and which will serve to illustrate this part of our subject. The peculiar attitude presented by the child offers as close a resemblance to experimental unilateral decerebration as is likely to be noted in man.

Case 11.—J. R., aged 2, was admitted to the Hospital for Sick Children, Great Ormond Street, on February 21, 1920, under the care of Dr. Poynton.

The mother's pregnancy was normal, and the child was born at full time by easy labour.

It was suspected in his second or third month of life he was not a normal child, because he was so quiet and inactive. He "lay like a doll" in bed or in his mother's arms. He was quite conscious, and took the breast readily, but moved neither legs nor arms, neither cried nor smiled, and apparently took no notice of his surroundings. About the age of 6 months he began to use his right arm, moving it about in a jerky way, and to draw up and kick his legs about. Since then there had been a gradual increase in the movements of the limbs, especially on the right. He had never been able to hold his head

FIG. 13 (Case 11).—Attitude approximating to that of experimental unilateral decerebration. The photograph was taken from above, the child lying on his back on the floor.

up, sit up, or stand, and had never learned to talk. During the last six months restless and jerky movements of the limbs became rather more obvious. There was no history of convulsions or fits at any time.

On admission the child was seen to be a healthy-looking little boy, of almost normal weight and length for his age. He seemed intelligent and bright, and followed movements with his eyes, but did not understand the simplest spoken word. He cried if subjected to pain, and if hungry or interrupted during a meal, but did not smile or respond in any way to

pleasurable stimuli, except to cease crying if he were already crying. He made no attempt to speak.

There was no true paralysis in any limbs, since the arms and legs could be moved in any direction, but their power was very feeble, and volitional control seemed extraordinarily erratic. The boy was unable to sit up, stand, or crawl, and if placed on his belly he could not turn over. There was a constant tendency to adopt a peculiar attitude (fig. 13); when lying on his back he practically always kept the right arm level with the shoulder in an abducted position, and flexed to a right angle at the elbow, the forearm midway between pronation and supination, the wrist somewhat over-extended and the fingers and thumb flexed; the left arm, on the contrary, was usually fully extended and close by the side or slightly abducted, forearm pronated and hand closed in a fist. The right leg was flexed at hip and knee, everted and abducted, with heel drawn up and toes pointing down; the left leg, on the other hand, was, like the homolateral upper limb, extended in full and sometimes slightly abducted. As a rule the head was turned to the right and somewhat retracted, chin tilted up and occiput lowered.

There was moderate tonus of the musculature; neither hypotonia nor rigidity or contractures were present.

In addition, however, frequent involuntary movements of a more or less choreiform character, jerky and apparently purposeless (objectively, at least), interrupted the maintenance of the posture above described. These were best seen when the child was held up by the axillæ, the limbs moving jerkily and erratically as in a Punch and Judy figure, the trunk wriggling and twisting and the head rolling restlessly.

Sensibility to pain appeared normal. All the deep reflexes were active; the abdominals were present and the plantars of the flexor variety.

The Wassermann test was negative in the blood and cerebrospinal fluid.

When we pass to what I am convinced are in fact one-limb or even unisegmental instances of the same decerebrate state the subject becomes still more interesting. Little, if any, attention has been paid to this aspect of the question, significant and informative though it is, although in 1872 Hughlings Jackson made a passing allusion to it in a few words: "In some cases of cerebellar disease there is only a fragment of the tetanus-like condition."

If we take, for example, the question of the head alone, neurologists are familiar with what has long been known as the "cerebellar attitude" of that part of the body. It is characterized, in unilateral lesions, by a tendency of the head to be tilted back and the occiput to be turned to the side of the lesion (fig. 14). To recall Sherrington's words in unilateral decerebrate rigidity: "The head is pulled towards the right (in right-sided lesions) and retracted." Now in complete or bilateral cases

it is to be supposed that the *turning* elements to the right and the left respectively will neutralize each other, whereas the *retracting* element, common to both, will result in the accentuation of that posture. And this is precisely what is found in complete decerebration: the head is strongly retracted in the mesial plane, and rotation is absent. This is the common position in bilateral diffuse basal meningitis and other organic states. Should one (right or left) mechanism for some reason predominate over the other the lateral-tilting element will be reintroduced.

FIG. 14.—Illustrates the "cerebellar" attitude of the head in a case of right cerebellar tumour. The typical decerebrate attitude of the head is compounded of two partly opposing, partly reinforcing, "cerebellar" components.

A similar explanation holds good for curving of the trunk (spinal column). The opisthotonos of bilateral decerebrate rigidity is compact of right and left *extending* elements, reinforcing each other, whereas the right and the left *rotating* elements neutralize each other. Occasionally, however, in not quite complete cases, the lateral deviating element can be detected, as in the figures in Sutherland and Paterson's forebrain aplasia cases. It will be remembered, further, that in the tonic fits of Case 7 lateral-rotating mechanisms were in full activity during part of the seizures. Probably there is a similar dissociation of "corresponding opposites" in the phenomenon of skew deviation.

Though, as remarked above, such head and other attitudes are commonly spoken of as "cerebellar," it has been shown that they are in reality part and parcel of the postures of decerebration; hence the term "cerebellar" is doubly misleading, since it fails to denote this integral association, and since, as will be shown, it is doubtful whether the origin of the attitudes is cerebellar at all. Clinically, in not a few cases of head deviation that I have seen, the lesion has been mesencephalic and the cerebellum quite intact.

FIG. 15 (Case 12).—A fragment of decerebrate posture. The left arm shows the pronation phenomenon in a case of tumour of the left edge of the middle lobe of the cerebellum.

To pass, in the next place, to instances of the condition being confined almost to a segment of a limb, or even to one phase of segmental movement, particular attention may be directed to the "pronator sign," which is an important part of the complete decerebrate posture. Nothing is more characteristic than this pronation or overpronation; it was noted by Sherrington in his experimental animals, and we have seen the frequency with which it occurs in human cases of cortical "ablation." In certain cases of limited lesion dissociating cortico-spinal and mesencephalo-spinal function unilateral pronation of the forearm has been the sole definite and persisting sign, a small but integral fragment of the complete decerebrate attitude, and therefore of considerable clinical significance.

FIG. 16 (Case 12).—Photograph taken from behind the patient's head. The pronator sign in the left arm is an integral part of decerebrate posture.

FIG. 17.—Pronation of the left forearm in a case of cerebellar disease, presumably in the left of the middle obe.

Case 12.—E. L., aged 12, had suffered for one year from the usual symptoms of intracranial tumour, viz., headache, giddiness, vomiting, and was found on examination to have well-marked double optic neuritis. Almost the sole localizing sign was a somewhat indefinite reeling gait, pointing, however, on the whole, to a lesion in the left cerebellum. Accordingly, the patient was trephined over this area. A great part of the left lateral lobe of the cerebellum was removed under the impression that it was gliomatous, but eventually a quite small encapsuled tumour, about the size of a cherry, was discovered at the left edge of the middle lobe. During the operation pronounced skew-deviation made its appearance, the left eye being down and in, and the right eye up and out. When the patient was being tested some ten days after the operation it was noticed that the left arm, when extended, always assumed an attitude (figs. 15, 16) of marked pronation and inversion—a symptom which had not been present before, and in addition the left leg was held extended and inverted at the foot. Since the operation, further, the head definitely assumed the posture of occiput to the left, chin to the right. A month later these postural attitudes were still maintained.

Fig. 17 is from a case of cerebellar disease exhibiting the same phenomenon as in Case 12. The presumption is that the lesion was in an identical spot, viz., the left side of the middle lobe; no opportunity of corroborating this, however, was afforded.

(3) Cases of Partial Decerebrate Attitude occurring as Involuntary Movements in the Course of Chorea and Athetosis.

Finally, I have for a long time been impressed with the way in which this pronator sign, a fragment of decerebrate posture, shows itself in cases of involuntary movement, more particularly in chorea and athetosis.

When the patient with acute or subacute Sydenham's chorea is asked to extend his arms above his head, palms to the front, it is extremely common to find them adopting involuntarily an attitude of pronation, with the palms turned outwards. The movement is well seen by contrast in cases of hemichorea (fig. 18), and is sometimes found persisting after the actual choreic movements have ceased. It is also to be observed in so-called paralytic chorea. Thus through the changing foreground of choreic twitching can be recognized a postural background of mesencephalic significance.

Similarly, in athetosis the pronator attitude is a position of election; it is, for example, vastly more frequent than any attitude of supination. Difficult though it be to follow the changing movements of athetosis,

Fig. 18.—Well marked pronator sign in a case of left unilateral Sydenham's chorea (hemichorea).

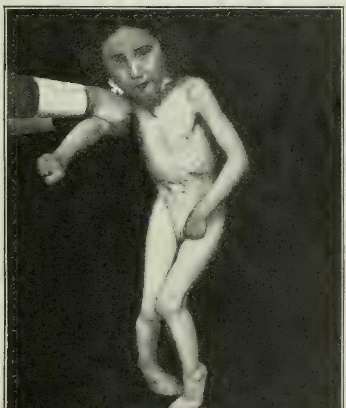

Fig. 19.—Characteristic transient decerebrate posture of right upper limb in a case of diplegia with athetosis.

there is, in my opinion, no doubt that the substantial background of that condition is one of varying decerebrate posture. Only one case need be cited in support of this contention; others are sufficiently illustrated in the photographs (figs. 19, 20).

FIG. 20.—Case of infantile cerebral hemiplegia with athetosis (left). Extreme hyper-pronation and characteristic decerebrate posture, with the exception that the fingers are extended and not flexed.

Case 13.—A. W., aged 10, developed diphtheria in January, 1919. The attack was of moderate severity. About fourteen days after the commencement of the illness, and while the patient was still very ill, she developed a complete left hemiplegia, involving face, arm and leg, without loss of consciousness and without any fit. Some weeks after the attack it was observed that the left arm was the seat of slow involuntary movements of an athetoid nature.

When the patient came under observation in June 1919, the hemiplegia was noted to be slight in degree, implicating mainly the left face and the left arm. Though the leg was not much complained of an extensor response was present. The left arm showed fair muscular power, though weaker than the right, and a fair range of voluntary movements, but these were interrupted by characteristic involuntary athetoid movements, more especially of the forearm, hand and fingers. There was a constant tendency for these movements to place the forearm in the attitude of over-pronation (fig. 21). Under treatment steady improvement took place, till at present the athetosis has very largely

disappeared; but the pronation-posture, though less frequently assumed, is still to be observed, especially if the patient is asked to extend her arms above her head.

In addition to the pronator sign it is possible to pick out others of the postural manifestations during the maintenance of decerebrate rigidity and observe their reproduction in the course of chorea and athetosis. One more phenomenon may be alluded to in this respect. It will have been noticed that in the great majority of the complete cases the arms are flexed at the wrists; Sherrington's experimental

FIG. 21 (Case 13).—Slight left hemiplegia with athetosis, following diphtheria. The left arm adopts an involuntary position of over-pronation.

animals showed the same flexion. Such wrist-flexion is, of course, the usual attitude at that joint in hemiplegia, representing, as has already been said, a fragment of decerebrate posture, and precisely the same flexion is extremely common in the choreic hand (fig. 22); it is always well seen when the arms are voluntarily extended in front. The choreic hand has long been known, but its significance in connection with decerebration does not appear to have been observed. Coupled with the flexion of the wrists is a tendency to finger over-

FIG. 22.—The choreic hand ; wrist flexion and some over-extension at the knuckles.

FIG. 23.—The athetoid hand, practically identical with the choreic hand (fig. 22).

extension, which is not observed generally in decerebrate rigidity (though in Case 8 the fingers were extended and adducted), but which, it must be understood, is largely voluntary in the test as usually employed, the patient being asked to "hold his hands out in front." This same flexion of the wrists is one of the commonest attitudes of athetosis (fig. 23).

There has recently been under my care at the National Hospital a case in which this combined wrist-flexion and finger-extension was a constant phenomenon in a child whose physiological condition was one not far from complete decerebration, and in whom the vestibular reflexes of Magnus and De Kleijn [14] were readily obtainable.

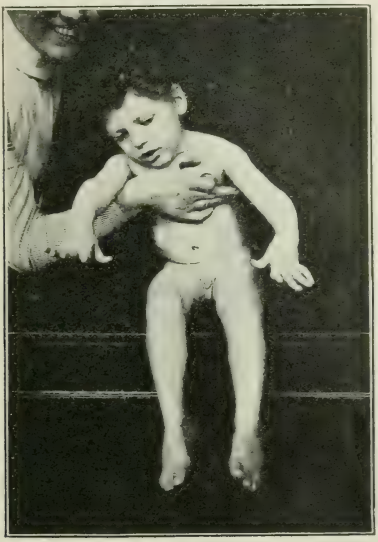

FIG. 24 (Case 14).—Microcephaly with diplegia and athetoid attitudes. For significance see text.

Case 14.—R. G., aged 3½, was admitted to the National Hospital under my care on June 5, 1919.

The infant was born after long labour with instrumental delivery. He was always backward, never tried to crawl, and could not use his arms properly. He never could hold his head up as other children.

On admission the child was noted to have a somewhat microcephalic head. He was extremely backward and infantile, though well enough developed

physically. Little if any attention seemed to be paid to interest taken in him. He wailed in a monotonous voice most of the time when left alone, but usually stopped when addressed, and occasionally an infantile smile crossed his features. The more or less constant attitude of the child is seen in fig. 24.

Magnus and De Kleijn reflexes.—When the child was placed on its back and the head gently turned, say to the left, and when it stopped crying, it lay quietly with upper limbs in a moderate flexion posture. On turning its head sharply to the right passively an immediate extension of the left arm into the usual decerebrate attitude was always obtained.

FIG. 25.—Torsion-spasm. The right arm is in a characteristic decerebrate posture.

One other variety of involuntary movement may be alluded to. In the rare disease known as torsion-spasm or torsion-dystonia (dystonia muscularum deformans) one or other limbs may adopt the decerebrate posture in its entirety. A striking case of this description under the care of my colleague Dr. Collier, who has given me every opportunity of examining it, has been fully reported by Dr. M. A. Blandy [2] and a photograph of the boy is here reproduced (fig. 25).

To recapitulate, thus far, in a sentence or two, the general view taken is as follows :—

(1) The symptoms of acute cases of physiological decerebration in men are identical with what obtains in animal experimentation. The decerebrate condition of forearm, wrist and fingers—extension at elbow,

pronation, flexion of wrist and usually of fingers—is commonly more marked than in animals, sometimes to an extreme degree. It is particularly noteworthy that respiratory disturbance often precedes or accompanies the decerebrate rigidity. The condition is frequently combined with tonic or postural fits, the feature of which is that they accentuate or reinforce the existing decerebrate attitude.

(2) Tonic or postural fits are, in reality, the transient adoption of this decerebrate attitude, complete or incomplete as the case may be. In the case of the fits there are changing phases of tonic attitude or posture which are less likely to be observed in the greater fixity of decerebrate posture produced by the release of certain neural mechanisms in structural nervous disease which *permanently* dissociate cortex from mesencephalon.

(3) The complete decerebrate attitude is compounded of unilateral elements which reinforce each other and of other unilateral elements which neutralize or antagonize each other. In the case of head and trunk postures, at least, it is possible to resolve them into components which, in incomplete cases, may preponderate on one or other side, producing deviations from the complete posture. This complete decerebrate posture is essentially composed of movements grouped in action in the mesial plane of the body.

(4) In cases where consciousness is intact and the corticospinal system partially or entirely in functional abeyance, portions of the decerebrate attitude make their appearance in the form of head, trunk, or limb deviations and attitudes; there may be fragments confined to one limb or segment of a limb. This is in particular the case with the pronator sign.

(5) In involuntary movements of the nature of chorea and athetosis it can be shown that there is a postural background which is composed of part of the complete decerebrate attitude. In both of these varieties the commonest phases are pronation and wrist flexion; the choreic and the athetoid hands are identical in this respect, and pronation and wrist flexion are an integral part of decerebrate posture.

(6) There is an important difference between the unconscious and the conscious cases, seeing that in the latter, unless the corticospinal interruption is absolute, the cortex may and often does interfere in part with the activity of the released lower mechanisms. In Sherrington's words, " there is a struggle between two conflicting influences."

The Pathogenesis of Decerebrate Rigidity.

We have seen that the resemblance between human and animal decerebrate rigidity is of the closest, and pathological evidence has been adduced indicating the nature and localization of the morbid processes likely to bring it about. From the type of pathological process, however, herein described as producing the syndrome (tumours, cerebral hæmorrhage, meningitis and internal hydrocephalus), it is apparent the localization can only be approximate, and physiological rather than anatomical; with other words, any lesion dissociating the cortex from the mesencephalo-cerebellar level and at the same time leaving the latter functionally intact, is calculated to cause the rigidity to develop, but the finer anatomical details can only be supplied at present from the experimental side. At the same time attention may again be directed to the fact that in the acute human cases marked disturbance of respiration has been repeatedly observed (see the cases in the first group, above). Graham Brown [10] has shown by exact experimentation that there is a respiratory tract, localizable with extreme nicety, in the mid-brain. In the ape it lies about 3 mm. from the aqueduct, and its stimulation produces gasping or panting movements. The occurrence of respiratory difficulty in the acute human cases may, perhaps, be attributed to interference with the function of this tract (assuming its presence in man) and thus provides important collateral evidence suggesting that the vicinity of the nucleus ruber (as is shown below), and not the cerebellum, is the neighbourhood whence is derived the activity that produces decerebrate rigidity.

The general physiological view enunciated by Hughlings Jackson [12] in 1877 as to the cause of hemiplegic rigidity is the starting point in our investigations of the pathogenesis of the symptom-complex under consideration. The following is his outline of the basic features of the "co-operation of antagonism" between cerebrum and cerebellum.

"There is evidently a duplex symptomatic condition, negative and positive; with loss of power over the muscles there is tonic action of them. Negative states of the nervous centres cannot *cause* positive states of muscles, they may *permit* them. My speculation is that the rigidity is owing to unantagonized influence of the cerebellum. . . . The cerebellum is the centre for continuous movements and the cerebrum for changing movements. . . . The former are tonic, the latter clonic. . . . In walking, the cerebellum preserves the equilibrium of the body, tends to "stiffen" all the muscles; the changing move-

ments of walking are the result of cerebral discharges overcoming in a particular and orderly way the otherwise continuous cerebellar influence. . . .

"When, however, the influence of the cerebrum is permanently taken off by disease of the cerebrum, as in hemiplegia, from the parts which it most specially governs, the cerebellar influence is no longer antagonized; there is unimpeded cerebellar influx, and hence rigidity of the muscles which in health the cerebrum chiefly innervates. The principle, I think, accounts for the duplex, negative and positive conditions in the contractures of hemiplegic children, in *the athetosis of Hammond*, and accounts for the rigidity and "propulsive" walk in paralysis agitans; temporary removal also for tetany and *certain hysterical attacks*, and also for some post-epileptic conditions of movements" (italics mine).

Specially noteworthy in this account is Jackson's recognition of "cerebellar influx" both in athetosis and in certain hysterical attacks; with characteristic prescience he sketched a hypothesis half a century ago, many of the details of which are only now receiving complete confirmation.

In regard to the above theory, however, the chief comment to be made is that the speculation as to the actual site of origin of the rigidity-producing influence being in the cerebellum cannot be considered proved, though the latter organ plays an important part. There is no question that the phenomena of decerebrate rigidity are release-phenomena, and that, again in Jackson's words, a lower influx invades what the higher gradually abandons.

For the determination of further essential facts in connexion with the subject of pathogenesis we must turn to the work of the experimentalists, conflicting though it is in some respects and not always of simplicity in interpretation.

In the first place, there is general agreement that decerebrate rigidity makes its appearance after transection of the neuraxis at the level of the anterior colliculi, and disappears by a second section below the neuronic level of the medulla. For its development, therefore, we are limited to the mesencephalon, cerebellum, and pons. It must, nevertheless, be mentioned there is experimental evidence suggesting that similar attitudes can be obtained when the decerebration is higher, leaving intact the basal ganglia, in particular the optic thalamus, and that movements analogous to those of tonic fits can be produced by stimulation of the latter structure (Lowenthal and Horsley [13],

Thiele [18], Graham Brown [8]). Rigidity, however, sets in with precision from mesencephalic transection, and for the moment we will confine consideration to the three parts of the neuraxis just specified.

This mesencephalic decerebrate rigidity, once established, can be abolished by various procedures. It will be readily understood that integrity of the lowest tone-effecting spinal proprioceptive arcs is assumed in this connection; with rigidity at its height, section of afferent roots at once abolishes rigidity for the limb concerned. Ordinarily speaking, therefore, for rigidity to appear a normal neuraxis below the mesencephalic level is presupposed, and if, clinically, a hæmorrhage spreads down the cord to invade local proprioceptive arcs the phenomena are certain to vanish. Thus the attitude of decerebrate rigidity may only be a phase in the course of a case and may disappear *sub finem*. Graham Brown [7], however, has made the important observation that integrity of these lower tone-effecting arcs is *not* absolutely essential. "The reactions (of decerebrate attitude) may occur many months after division of the dorsal spinal roots of the arm. That is to say, appropriate stimulation in the region of the midbrain may evoke an extensor postural tonus or a flexor postural tonus. Sherrington has found that the decerebrate rigidity which occurs after removal of the cerebrum does not occur in a 'de-afferented' limb, but the fact that a condition which at any rate very closely resembles this state may be evoked in such limbs seems to point to the conclusion that the absence of this postural tonus in the decerebrate 'de-afferented' animal is due to the failure of the ascending impulses from the limb which normally play—however indirectly—upon these mechanisms of the midbrain, and that the mechanisms themselves if properly activated are still able to induce the tonus."

In addition to the local spinal reflex arcs for tone, it is held by Sherrington, as Graham Brown says in the above citation, that impulses ascend to the midbrain mechanisms to maintain them in activity, but they do not pass by the dorsal columns, for section of these has no effect, whereas section of the ventro-lateral cord abolishes the rigidity. This suggests the conveyance of maintenance-impulses by the spino-cerebellar tracts, and Weed [20] believes the path is the ventral spinocerebellar tract of Gowers, and not the dorsal, since section of the inferior cerebellar peduncles, by which the dorsal pass to the cerebellum, is without influence on established rigidity.

Another method of abolishing the extensor tonus after decerebration is by excitation of the anterior (superior) surface of the cerebellum,

from near the midline outwards to near the lateral border of the cerebellar surface (superior vermis and outward). This has been observed repeatedly by Sherrington, Lowenthal and Horsley, Weed and others. If, then, stimulation of this part of the cerebellum inhibits an induced decerebrate rigidity, it might be supposed, conversely, that destruction of the same area would produce the condition, mesencephalic decerebration not being performed, and this has been done by Sherrington and by Thiele. Bisection of or injury to the vermis, median section of the cerebellum, and partial ablation of the cerebellum, are all apt to produce the phenomena under discussion, but Sherrington is not convinced that the two conditions are identical, and specially states that after these cerebellar operations decerebrate rigidity "sets in often, *but not always*" (italics mine). He has seen the condition "persist after removal of the cerebellum, if the latter ablation be performed without any serious amount of hæmorrhage." Graham Brown, similarly, has clearly shown that the reactions of decerebrate rigidity may be obtained from stimulation of the midbrain "after the complete removal of the cerebellum"; and Thiele has removed the whole cerebellum down to the paracerebellar nuclei (Deiters) without affecting the decerebrate posture.

It is apparent, therefore, that functional integrity of the cerebellum is *not* a *conditio sine quá non* for the appearance of the syndrome after mesencephalic transection in animals, and that the phenomena may develop in the actual absence of that organ.

These experimental findings seem to render an actual cerebellar origin for the rigidities improbable.

The position reached by Sherrington, Weed, and Graham Brown is that the mesencephalon itself is of prime importance. According to Weed, the real origin of decerebrate rigidity is in the midbrain in the region of the colliculi. "In this portion of the midbrain, lying ventral to the colliculi, the tegmental structure which apparently affords origin for the rigidities is the nucleus ruber." Graham Brown has developed the hypothesis in greater detail and with a wealth of experimental material. He has shown that "the red nucleus is the centre the activity of which conditions" the postural reflexes constituting decerebrate rigidity. And in a personal communication to the writer Professor Sherrington says:—

"The neighbourhood of the iter taken broadly so as to include the red nucleus, small-celled and large-celled parts, certainly has large postural (tonic) functions, and Graham Brown's experiments observed those in detail. Work on decerebrate rigidity has gradually come to

suggest that the red nucleus region is of capital importance for the general reflex of 'standing' ('standing' in its various forms and sub-postures). Tonic (posture) fits would, to judge from laboratory observations, be likely to result from lesions of an acute kind in that region."

From the point of view of experimentation, then, the conclusion seems clear enough that the withdrawal of cortical control, by decerebration, from the spinal motor centres allows them to be played on by a non-pyramidal motor influence of a tonic nature, or rather allows this doubtlessly constant tonizing factor to reveal itself in action. The centre of this attitude- or posture-producing motor activity is apparently the nucleus ruber of the mesencephalon, and not the cerebellar cortex or cerebellar nuclei, and it is maintained in tone itself, ordinarily, by afferent impulses reaching it, "however indirectly," from lower (spinal) neural levels, though its activities are not necessarily paralysed by their absence.

The nucleus ruber, however, is no isolated and autonomous ganglion, but has numerous and important connections with structures above, beside, and below it. Further, and of equal importance, it is a twin nucleus. We cannot suppose, though it be the originator of decerebrate rigidity, that it is uninfluenced by stimuli from parts with which it is in close relation.

(1) Though the postural discharges of decerebrate attitude have not their origin in the cerebellum, the latter must certainly play a part, as long, that is, as it is in functional connection with the midbrain. We have already seen that the rigidity can be inhibited by excitation of the neighbourhood of the vermis, while lesions there would appear themselves to be provocative of rigidity, the mesencephalon being intact, or to accentuate existing rigidity. This experimental finding is supported by the clinical evidence of Case 12 above, since the destructive lesion allowing the "fragments" of decerebrate posture (pronation) to appear was situated at the left edge of the middle lobe, i.e., in the position whence decerebrate rigidity can be produced experimentally by destructive lesions. Thus the cerebellum would appear to exercise both an inhibitory and an excitatory action on the mesencephalic mechanism which is responsible for decerebrate posture. According to Weed, who produced the condition by removal of the cerebral hemispheres, leaving the internal capsules and basal ganglia (if I understand his descriptions correctly), it is possible to trace an inhibitory pathway from the cerebrum which corresponds to the fronto-

ponto-cerebellar route (viâ the middle cerebellar peduncle). Stimulation of this pathway always inhibited existing decerebrate posture, mainly homolaterally, suggesting a doubly crossed connection. Weed found the path is distributed, in part at least, to the cortex of the anterior portion of the superior vermis, whence, as has been said, previous experimenters have been able to inhibit the rigidity, but whether the inhibitory effect is exercised upon the basal cerebellar nuclei (Deiters) or upon the mesencephalic nuclei is not at present known.

As far, then, as cerebellar action is concerned, it may be tentatively concluded that (1) decerebrate attitude of mesencephalic origin is maintained by afferents reaching its source by spinocerebellar paths, and (2) inhibited by stimulation of a ponto-cerebellar path viâ the neighbourhood of the vermis, i.e., of what constitutes largely the palæocerebellum. Possibly the maintaining influence reaches the twin red nuclei by the superior cerebellar peduncles, since their section, in the decerebrate preparation, is usually followed by disappearance of the rigidity; as already noted, it is not certain how the inhibiting influence is conveyed to the mesencephalon, or even whether it may not pass back again to spinal centres by another route.

(2) The basal ganglia have intimate connections with the red nucleus. In particular, the corpus striatum is in relation with it by means of the ansa lenticularis. As our subject is mainly concerned with the phenomena appearing after transection through the midbrain, when the basal ganglia are out of action, it is not necessary to discuss the physiological connections of the two in this place, though several allusions have already been made to the fact that with suitable experimentation the rubral mechanism of posture can be activated from above, from the basal ganglia. In a subsequent communication concerned with the pathogenesis of involuntary movements further opportunity will be taken to analyse the bearing of the influence of the basal ganglia on mesencephalic function, and a brief reference to the connection of the red nucleus with tremor is given below.

We may turn next to the question of the rigidity-producing mechanism itself.

The careful work of Graham Brown has shown that in the decerebrate preparation electrical stimulation of certain points in the cross-section of the midbrain at the level of the anterior colliculi in monkeys yields characteristic reactions. The areas excited, his "focal points," lie on either side of the midline some millimetres ventral to the aqueduct

of Sylvius, and correspond with the anatomical position of the red nucleus (and possibly, also, with that of the posterior longitudinal bundle). "Stimulation applied to this area of the cross-section of the midbrain on one side evokes a state of flexion in the arm of the same side and a state of extension in the arm of the opposite side. On cessation of stimulation these reactions are continued as maintained postures. The movements of the arms are accompanied by movements of other parts of the body. The tail seems to bend to the same side; the lower limb of the same side extends while that of the opposite side flexes; the head is rotated in such a manner that the face is turned away from the side stimulated." He has shown, further, the mutual antagonism of the right and left focal points, and by immediate or successive compounding of the two has produced bilateral extension, bilateral flexion, and so on. "With varying values of stimulation varying values of contralateral extension and ipsilateral flexion might compound to give reactions in which extension and flexion exhibited differing preponderancies."

Now our clinical study has already demonstrated the fact that the clinical phenomena of decerebrate rigidity are compounded of elements some of which are mutually reinforcing and others mutually antagonistic, and in Graham Brown's work we have experimental proof of the way in which these symptoms are produced. It is evident that the varieties and alternations of positions—e.g., all limbs extended, as is usual in human decerebrate rigidity, or arms flexed and legs extended as in Jackson's "tetanus-like seizures"—are due to compounding of varying influences affecting the red nuclei or originating in them. And it seems no long step to the supposition that unisegmental attitude, as, for instance, in the pronator sign, is the result of limited or partial red-nuclear activity.

It may be suggested that in the red nucleus, perhaps in the pars magnocellularis, are represented combinations of the ventral cell-groups of the spinal cord, linked up according to their flexor, extensor, pronator, supinator, &c., functions. For we cannot believe that in the red nucleus we have other than a "re-presentation" of the ventral cord cell groups. Graham Brown has, indeed, demonstrated that the point of common antagonism between the twin nuclei is below the midbrain, and the natural surmise is that it lies at as low a level as that of the spinal centres. If this be the case, then stimulation of a point in the red nucleus, after decerebration, may be supposed to innervate a physiological group of ventral cells, the flexors, say, scattered throughout the

various spinal segments, and all linked to a nodal cellular representative in the former nucleus. The same must be true of extensor cells. &c. Uncontrolled activity of the twin nuclei from decerebration throws the individual into the decerebrate posture, with predominance or changing activity of flexor or extensor attitude on one or other side, in one or other limb, as the case may be, owing to changing influences in the red nuclei or reaching them.

Undoubtedly the predominating activity of the uncontrolled nuclei is one causing extensor tonus. The "neural balance" of a limb-pair, taken together, seems for some reason to be dominantly weighted or disposed towards, or "set in," extension [9]. Sherrington's explanation of this is that decerebrate posture is in reality "reflex standing," hence the preponderance of extensor tone.

Throughout this discussion, for the sake of clearness, the assumption has to some extent been made that it is precisely the red nucleus in which these postural activities are anatomically centred, though greater certainty is necessary before the matter can be regarded as settled. But it seems desirable to point out that the nucleus ruber is but a part of Edinger's nucleus motorius tegmenti, which consists of associated motor-cell groups scattered through the pons and mesencephalon to the level of the ventral optic thalamus, including Deiters' nucleus, and that the effect of stimulation is not likely to be limited to one descending rubral path, viz., the rubrospinal. There is, in fact, doubt as to the path or paths by which rubral activities reach the cell-groups of the ventral cornua; further, rubro-pontine and rubro-bulbar connections must not be ignored. In this connection it is well to mention that Thiele [18] attributes the conduction of decerebrate impulses spinalwards, not to the nucleus ruber and its connections, but to Deiters' nucleus, another part of the nucleus motorius tegmenti, and to the vestibulospinal tract. Thus there is room for further research and elucidation in the matter. It is clear, for example, that other influences reach the final common path of the lower motor neuron than the rubrospinal. The possibility of labyrinthine agency contributing a quota to decerebrate rigidity, originally mooted by Ewald, has been largely supported recently by the researches of Magnus and de Kleijn [14], who have proved that in the decerebrate animal passive movements of the head give rise to alterations of tonus in the limbs, and that each movement of the head varies in its influence on tonus according to the position of the animal in space. Only this passing allusion to their investigations can be now made.

Although the question is one to be dealt with elsewhere, brief

mention may be made, in conclusion, of the connection of the red nucleus with involuntary movement. If the hypothesis suggested above be tenable, viz., that the flexor, extensor, &c., groups of the spinal cells are respectively linked to nodal representatives in the nucleus—are, as it were, re-assembled in these twin ganglia—then we can understand how tremor may occur in association with rubral disorder. For, in Jackson's words once more, "tremor differs from rigidity, not fundamentally, but in degree."

REFERENCES.

[1] Bastian. "A Case of Apoplexy in a Boy aged 15: Intraventricular Hæmorrhage, Convulsions, and Death in four hours," *Trans. Clin. Soc.*, 1884, vol. xvii, p. 21.
[2] Blandy. "A Case of Torsion-dystonia, or Torsion-spasm," *Journ. of Neur. and Psych.*, 1920, vol. i, p. 148.
[3] Bruce and Drummond. "A Case of Intracranial Aneurism in a Young Subject," *Rev. of Neur. and Psych.*, 1904, vol. ii, p. 737.
[4] Bruce, Pirie, and Macdonald. "Aneurism of the Anterior Cerebral Artery, with unusual Prolongation of Life after Rupture; Autopsy," *Rev. of Neur. and Psych.*, 1908, vol. vi, p. 449.
[5] Eskridge. "Tumour and Large Cyst of the Cerebellum, with Symptoms extending over several Years," *Med. Record*, August 17, 1895.
[6] Firth. "Cerebellar Fits of the Hughlings Jackson type," *Journ. of Neur. and Psych.*, 1920, vol. i, p. 48.
[7] Graham Brown. "On Postural and Non-postural Activities of the Mid-brain," *Proc. Roy. Soc.*, B, 1913, vol. lxxxvii, p. 145.
[8] *Idem.* "On the Effect of Artificial Stimulation of the Red Nucleus in the Anthropoid Ape," *Journ. of Physiol.*, 1915, vol. xlix, p. 185.
[9] *Idem.* "The Rule of Reflex Response in the Limb Reflexes of the Mammal and its exceptions," *Journ. of Physiol.*, 1912, vol. xliv, p. 125.
[10] *Idem.* "A Respiratory Tract in the Mid-brain," *Proc. Physiol. Soc.*, May 16, 1914.
[11] Hughlings Jackson. "Case of Tumour of the Middle Lobe of the Cerebellum: Cerebellar Paralysis with Rigidity (cerebellar attitude); occasional Tetanus-like Seizures," *Brain*, 1906, vol. xxix, p. 425.
[12] *Idem. Med. Examiner*, 1877, April 5, p. 271.
[13] Lowenthal and Horsley. "On the Relation between the Cerebellar and other Centres, &c.," *Proc. Roy. Soc.*, 1897, lxi, p. 20.
[14] Magnus and De Kleijn. "Die Abhängigkeit des Tonus der Extremitätenmuskeln von der Kopfstellung," *Pflüger's Archiv*, 1912, Bd. cxlv, S. 455.
[15] Sherrington. "Decerebrate Rigidity and Reflex Co-ordination of Movements," *Journ. of Physiol.*, 1897, vol. xxii, p. 319.
[16] Stewart and Holmes. "Symptomatology of Cerebellar Tumours," *Brain*, 1904, vol. xxvii, p. 543.
[17] Sutherland and Paterson. "A Type of Cerebral Maldevelopment (Forebrain Aplasia)," *Quart. Journ. Med.*, 1915.
[18] Thiele. *Proc. Roy. Soc.*, 1905, vol. lxxvi, p. 360.
[19] Turner, Violet. "A Case of Prolonged Hyperpyrexia in a Child with a Mid-brain Tumour," *Brit. Journ. of Child. Dis.*, 1916, vol. xiii, p. 261.
[20] Weed. "Observations on Decerebrate Rigidity," *Journ. of Physiol.*, 1914, vol. xlviii, p. 205.

RELATION OF THE FORM OF THE KNEE-JERK AND PATELLAR CLONUS TO MUSCLE TONUS.[1]

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I.—INTRODUCTION.

VARIATIONS in the form of the knee-jerk under various degrees of muscle tonus have been observed both clinically and in the physiological laboratory. These observations have extended over many years from the clinical deductions of Erb and Westphal in 1875, through the more recent physiological experiments of Sherrington and others. From these studies the clinician has drawn certain practical concepts such as "exaggerated knee-jerks," "spastic knee-jerks" and "spasticity," terms used to denote the stiffness of the muscle and the increase in the amplitude of the reflex response most commonly seen after lesions of the pyramidal tracts. These expressions are used in a rather loose way, simply denoting gross quantitative changes, for the most part not to be measured with much degree of accuracy.

Attempts, however, have been made to measure and record variations of the knee-jerk in man and to show the relations between these variations and muscle tonus. Weir Mitchell and Lewis [11] in 1886 contributed the first of such observations and came to the conclusion that "the responsive jerk brought about by striking a stretched tendon is the most refined means we possess of deciding as to the tone of a muscle." This, I think, has been borne out by research since that date, although we have developed more "refined means" to aid in the decision. Bowditch and Warren [1] in the Harvard Laboratory two years later measured the knee-jerk in man by a cleverly arranged but complicated apparatus. Lombard [4] also carried out experiments on man. Somewhat later Wertheim Salomonson [12] published a paper on reflex studies in man with kymographic records. It was noted by all experimenters on men that numerous difficulties prevented the correct recording of their observations. Most of these workers found great mechanical difficulty in attaching a steady apparatus to the lower

[1] From the Physiological Laboratory, Oxford University.

extremity. Voluntary movements of the patient or his voluntary inhibition of the reflex also caused great variations in their result. These latter phenomena could not be successfully controlled. In short, all attempts to study any single phase of the knee-jerk in man, such as its form, and to record the observations graphically have been complicated by so many factors which could not be controlled that results from such studies are only partially satisfactory.

In animal experiments, however, many of the extraneous factors mentioned above can be controlled by the experimenter. Unfortunately, except for the work of Sherrington, there have been few detailed studies in animals on the relation of the form of the knee-jerk to muscle tonus. It was the aim of this research, then, to demonstrate in animals just what form the knee-jerk may assume under various degrees of tonus. We only attempt to isolate the knee-jerk nerve-muscle group in a living preparation and study it under various degrees of muscle tonus.

II.—Method.

(1) *Preparation of animal and apparatus.*—For a study of the knee-jerk in an animal the nerve-muscle preparation isolated was the anterior crural-vasticrureus group. This was comparatively easy in the cat. The dissection as described by Sherrington [7] cuts all the muscles and nerves of the hip and thigh except the internal and external vasti and crureus muscles and the anterior crural nerve connecting them with the central nervous system. The proximal end of the peroneal nerve was also left for external stimulation. This dissection was usually bilateral, giving two knee-jerk preparations. In all cases both peroneal nerves were isolated. Thus we had an isolated reflex group, free from the influence of muscles acting on the same joint which might interfere with the recording of movement of that joint.

Cats were used for all experiments. The preliminary dissections described above to isolate the knee-jerk muscles were done under ether anæsthesia. The two peroneal nerves were exposed at the same time and glass-shielded electrodes attached to their proximal ends. A faradic current was used for stimulation of these nerves. The feet of the animals were amputated at the ankles and a pin attached firmly through the tendons and periosteum at the distal end of the tibiæ to provide a point of attachment for our recording apparatus. If a spinal preparation was to be used a preliminary laminectomy was done at

this stage, exposing the cord without opening the dura at about the tenth dorsal vertebral level. The skin was used to cover these operative defects so as to keep the limbs and back warm. All experiments were also performed on a warm table.

After the above steps, decerebration was carried out by using the Sherrington decerebrator. In practically every case decerebration was successful and good decerebrate rigidity was obtained. The animal was then placed on its back surrounded with hot-water bottles to insure a fairly even temperature. The head was slightly elevated. Animals could be kept in good condition for hours, in one case for over twenty-four hours. The legs were fixed by pivot pins through holes drilled in the condylar aspects of the femurs. A thread from the fixed pin at the ankle passed over a series of pulleys to recording levers, making a mark on a smoked kymographic drum. An upward fling of the leg (extension of the leg by contraction of the vasticrureus muscle), when the patellar tendon was tapped, recorded on the drum as an upward vertical mark. Variations in the tone of the muscle were recorded on the drum by the various levels of the marker. Thus a low level on the drum indicated low tonus (a low-tension muscle) [9], while a high level denoted a high tonus (high-tension muscle). Electrodes of the Sherrington [6] bipolar type were then attached to both peroneal nerves by separate circuits for stimulation by the faradic coil.

If a spinal preparation was desired the previously exposed spinal cord was cut entirely through at the tenth dorsal level, and the animal was then turned on its back and the same system of recording the movements of the vasticrureus as above described was used.

(2) *Variation of tonus in the isolated reflex group.*—Sherrington [8] had found that there was a marked increase in tonus after decerebration in extensor muscles such as the vasticrureus. This tonus of an antigravity muscle group can be decreased or increased by the stimulation of a sensory nerve of the same side or opposite side of the body. Thus, if the peroneal (partly sensory) nerve, which is convenient and accessible, be stimulated in the ipsilateral limb the tonus of the vasticrureus group is at once lowered. Vice versa, if the contralateral peroneal nerve be stimulated the tonus of this antigravity muscle group is increased. The relaxation of the extensor vasticrureus when an ipsilateral nerve is stimulated is part of the reciprocal innervation of the flexion reflex. Thus stimulation of ipsilateral and contralateral peripheral nerves enabled us to vary the tonus of this extensor muscle. By cutting the spinal cord in the mid-thoracic region we had a spinal

preparation with flaccid limbs and "toneless" muscles. Thus we could vary our tonus from complete rigidity, sometimes seen in the freshly decerebrate preparations, to flaccidity seen in the spinal animal, with variations between these two extremes by stimulation of the ipsilateral or contralateral peroneal nerves.

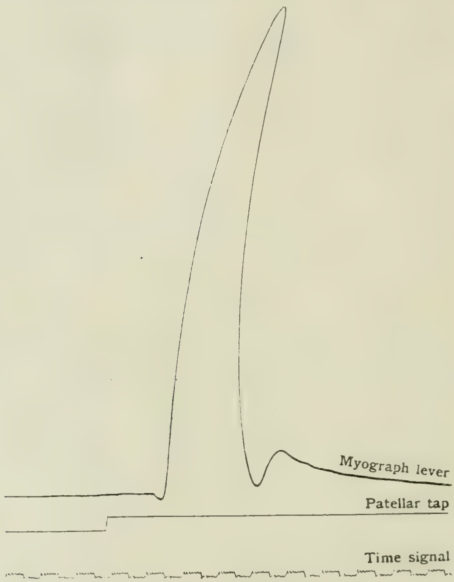

FIG. 1.—Myogram of the form of the isolated knee-jerk of a spinal cat. The upper line records the movement of the vasticrureus muscle when the patellar tendon is tapped. An upstroke indicates contraction of the muscle; a downstroke, relaxation. The approximate time of the blow of the patellar is indicated by the second line. The lower line is the record of a tuning fork vibrating 13·5 times per second.

III.—OBSERVATIONS.

(1) *The knee-jerk in the spinal and decerebrate cat.*—The response to a tap of the patellar tendon in a spinal preparation was first examined. The spinal cord had been cut through at the level of the tenth dorsal segment and the vasticrureus muscle showed very little tone. When the limb was elevated and let fall it would drop back to its original position. There was no "lengthening" or "shortening"

of the muscle on passive movement. After any manipulation the same level as before was recorded on the drum (i.e., the tension of the muscle remained constant). Although the muscle was flaccid to palpation the knee-jerk was fairly active. A unilateral record taken of this preparation is shown in fig. 1. The curve is a simple one of contraction followed by an almost parallel relaxation, the same level being maintained after the jerk as before. There was a slight rebound after the jerk. This rebound was thought not to be due to either mechanical bouncing of the knee-joint or the lever. It was presumed that the sudden elongation of the muscle after its contraction was enough of a stimulus itself to give another knee-jerk. With the cat on its back and the leg only supported by the distal end of the femur, the weight of the leg is, of course, a large factor in its return to its original position after the jerk. The second line of the record indicates the moment a hammer fell from its fixed position just above the patellar tendon. It does not, therefore, represent the exact moment the tendon was hit, for a slight amount of time was taken by the hammer falling a few centimetres. This record cannot be used, therefore, to measure the time of the reflex, as it gives approximately only the exact time of the stimulation.

Fig. 2 is a record of the same decerebrate animal without the spinal cord cut. In this experiment there was a moderate degree of decerebrate rigidity. All the extensor muscles of the upper limb were tonic and the vasticrureus was spastic to palpation. After the limb was extended from a flexed position by hand the muscle continued to stay shortened and showed a higher level on the drum than before it was moved. Vice versa, if the limb was flexed by hand a new level of tone was developed which continued without further change. The limb, therefore, showed the "shortening" and "lengthening" reaction and "plasticity," terms used by Sherrington to describe these phenomena; in short, the vasticrureus muscle could be "set" at various lengths and it would remain fixed at its new length. This plastic element in these spastic muscles was demonstrated in many decerebrate preparations, and we came to recognize it as one of the most constant signs in decerebrate rigidity. In this spastic vasticrureus muscle the knee-jerk showed a form quite different from the non-spastic or spinal muscle. Two elements would be easily recognized: the jerk which appeared first and resembled the jerk of the spinal knee-jerk, and the second factor, the plasticity. The jerk was slightly greater in amplitude than the corresponding jerk in the spinal animal. After the jerk, however,

the muscle did not completely relax but remained partly contracted at a shorter length than before it was stimulated. This shortening showed as a higher level on the recording drum. Thus there was a new tonic level after each knee-jerk more tonic than the level before the jerk. The muscle continued at this level or tonic state until another jerk or

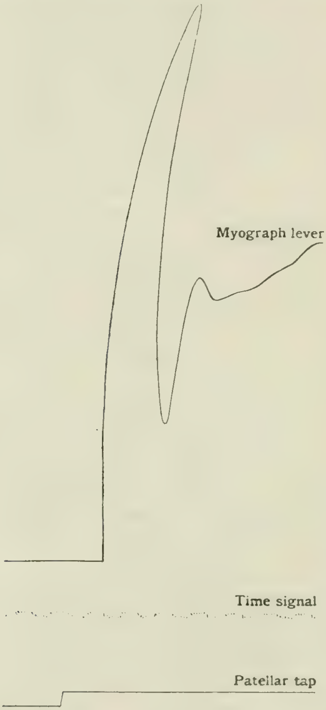

FIG. 2.—Myogram of the form of the isolated knee-jerk of a decerebrate cat. The tracings indicate movements as in fig. 1, except that the time record is placed above that of the patellar tap.

some other factor changed its tonus. The form of the knee-jerk, therefore, was changed in the decerebrate from the simple jerk seen in the spinal animal to a jerk plus a continued shortening of the muscle, the second element being the result of the "plastic tonus." Fig. 2 is a typical curve taken from many, and when compared with fig. 1 the

difference in the final phase of the knee-jerk is apparent. In the cat, then, we may say that increased tonus of the vasticrureus muscle (decerebrate) causes a change in the form of the knee-jerk, the jerk being slightly greater in amplitude, and also exhibiting plasticity not found in the muscles with slight tonus (spinal).

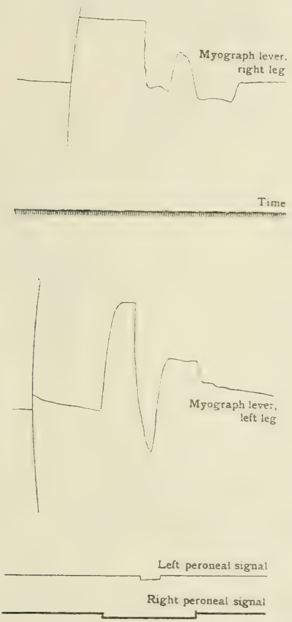

FIG. 3.—Reciprocal innervation in two isolated vasticrurei muscles. (See text for description.) An upstroke of the lever indicates increased tension of the muscles; a downstroke, decreased tension. Time, 13·5 beats per second.

We may vary the tonus of the vasticrureus muscle by stimulating the ipsilateral or contralateral peroneal nerves. Fig. 3 is a record of such an experiment. The movements of the two vasticrureus muscles are recorded as well as the time of the peroneal stimulations. The first nerve stimulated was the right peroneal. The tonus of the right extensor muscle was inhibited by this stimulation and the tension of the muscle relaxed, i.e., the writing point dropped; at the same time

the tone of the left vasticrureus muscle was increased, the tension increased and its lever was raised. While the tone of the right extensor muscle was lowered and that of the left raised, we stimulated the left peroneal nerve and the reverse of the above took place; the tonus in the right muscle was increased, and in the left muscle

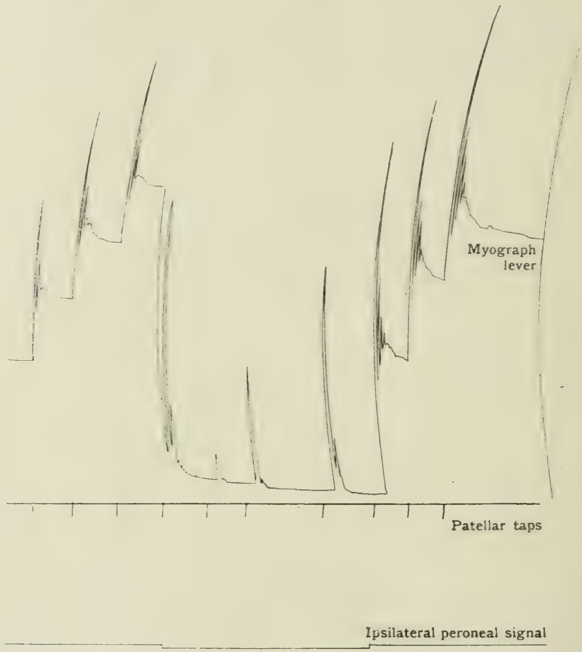

FIG. 4.—Showing the effect of ipsilateral peroneal stimulation on the form of the knee-jerk in a decerebrate cat. (See text for description.)

decreased. As the stimulation of these two peroneal nerves was removed by cutting off the faradic current in each, the original movements described above took place in the reverse order. It is clear, then, that we were able to raise or lower the tonus of these knee-jerk muscles by the use of contralateral or ipsilateral peripheral stimulation.

(2) *The knee-jerk in tonic variations.*—Fig. 4 illustrates an experi-

ment in a decerebrate preparation showing marked spasticity with the usual plastic element. The first three knee-jerks recorded show the increase of tonus after each jerk. This causes a "staircase" effect in the record, a condition characteristic of decerebrate rigidity in the extensor muscles of the cat. The tone of this extensor muscle was then lowered by stimulation of the ipsilateral peroneal nerve, and during this stimulation a number of knee-jerks were elicited corresponding to the spinal type of knee-jerk in each case. Although they varied somewhat in amplitude the relaxation after the jerk was as great as the

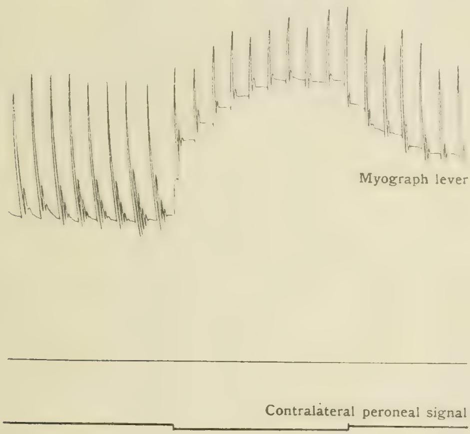

FIG. 5.—Showing the effect of contralateral peroneal stimulation on the form of the knee-jerk in a spinal cat. (See text for description.) Points of patellar taps not indicated.

contraction and the element of plasticity in the muscle has been eliminated. After the current was released the knee-jerk again assumed the form of the decerebrate preparation. Of the ten knee-jerks recorded in this figure the first three and the last three show the typical decerebrate form with plasticity, while the four middle ones show the simple twitch or spinal type. The middle line of the record marks approximately the time of stimulation of the patellar tendon, while the lower line indicates the beginning and ending of the ipsilateral peroneal stimulation. There was a slight tendency to clonus (pseudo-clonus) in all of the decerebrate knee-jerks, not so marked in the spinal ones.

As fig. 4 represents a spastic or decerebrate knee-jerk taking the

form of a flaccid or spinal knee-jerk by inhibition of the tonus, fig. 5 shows the reverse condition, a flaccid knee-jerk of a spinal animal becoming a spastic knee-jerk as in a decerebrate animal. This record

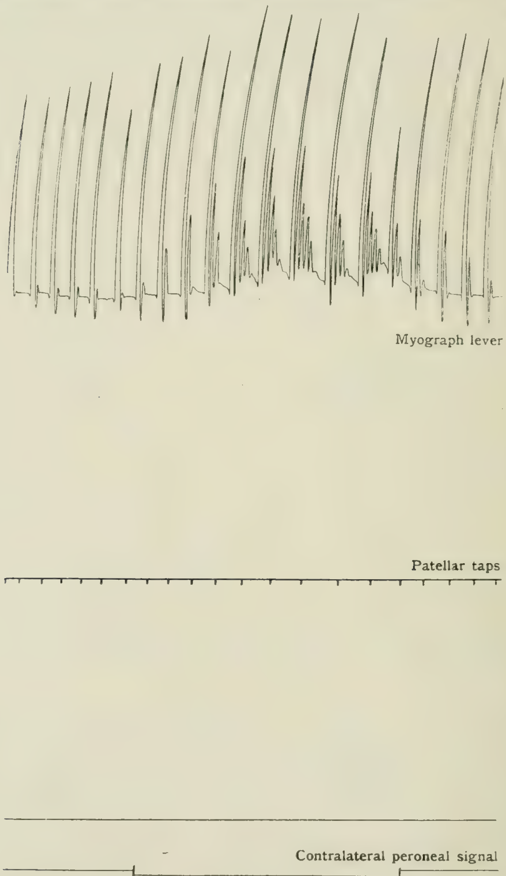

FIG. 6.—Slight increase in tonus with contralateral peroneal stimulation in a spinal cat with development of a pseudoclonus. Middle line indicates points of patellar taps. Lowest line shows point of peroneal stimulation.

is of an experiment on a spinal animal, and it can be seen that the knee-jerks at the beginning of the record are typical of the spinal type. Between the ninth and tenth knee-jerks on the record the contra-

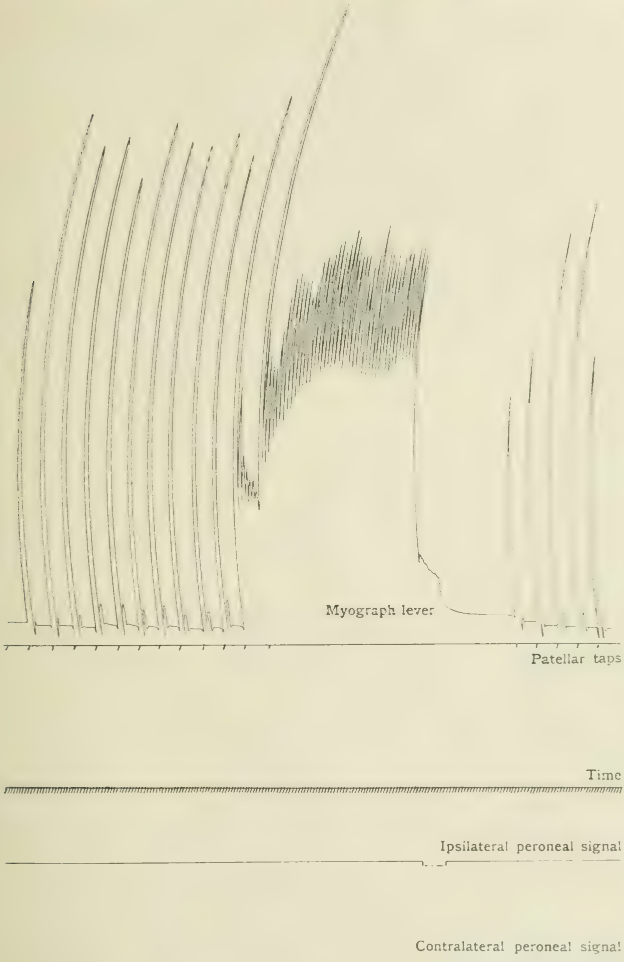

FIG. 7.—Knee-jerks in spinal cat augmented to a clonus by contralateral peroneal stimulation and stopped by ipsilateral inhibition. The first ten knee-jerks are "spinal" in form, At this point contralateral stimulation raised the tonus, and the next knee-jerk was spastic. followed by another with clonus. The tonus was then strongly inhibited by ipsilateral stimulation, the clonus stopped and the knee jerks resumed their spinal form. The upper line is the myograph lever, below this, the signal of the patellar taps, the time 13·5 beats per second, ipsilateral peroneal signal, and below it, contralateral peroneal signal.

lateral, peroneal nerve was stimulated, thus increasing the tonus of the extensor muscle, and during this increase of tone the knee-jerks assumed the spastic form with spasticity. When the stimulation was released, the knee-jerks again resumed their spinal character. The lowest line of the record indicated the beginning and ending of the contralateral peroneal stimulation.

In the last two records, therefore, we have first a preparation with a high degree of tonus and plasticity showing the typical decerebrate knee-jerks reduced by ipsilateral stimulation to a preparation with a low degree of tonicity showing the spinal type of knee-jerk. On the other hand, the second experiment illustrates the exact reverse of this; a spinal animal with a typical spinal knee-jerk has its tonus in the extensor muscle increased by contralateral stimulation, and the knee-jerks show the decerebrate or spastic type while the muscle is thus stimulated.

(3) *Patellar clonus.*—A pseudoclonus was often noted in both the spinal and decerebrate preparations. There was more tendency for it to develop when the tonus was slightly elevated by stimulation of the proximal end of the contralateral peroneal nerve (fig. 6). It will be seen that the knee-jerk showed a slight degree of plasticity during this stimulation, and also that the "rebound" or second jerk became a short series of jerks from two to four in number. If, however, the tonus augmenting stimulus was slightly stronger than shown in fig. 6, a real self-sustaining clonus could be developed, the relaxation after each contraction being enough of a stimulus presumably to set off another jerk (fig. 7). During this experiment, when the tonus was suddenly dropped by an ipsilateral peroneal stimulation, the clonus at once stopped and the knee-jerk resumed its original spinal form.

Such a clonus was often started in a decerebrate animal with a spastic vasticrureus muscle by raising the tonus by the "staircase" method of repeated knee-jerks. Fig. 8 is the record of an experiment showing that one patellar tap was sufficient to cause a pseudoclonus and raise the tonus enough so that the second tap was followed by a continued or actual clonus.

Fig. 9 illustrates an experiment in which the clonus is shown in a little more detail. This tracing, from an experiment on a decerebrate cat with a spastic vasticrureus muscle, illustrates the development of a clonus after a single knee-jerk raised the tonus to a certain level. It also illustrates the regularity of the contraction and relaxation of the muscle during the clonus. The rate of the clonus in

FIG. 8.—Clonus in vasticrureus muscle of decerebrate cat inaugurated by successive knee-jerks, the "staircase" effect. The middle line is the signal for patellar taps. The lowest line is the time record, 13·5 beats per second.

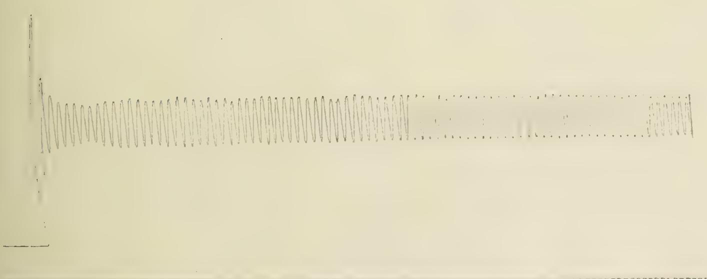

FIG. 9.—Patellar clonus in decerebrate cat, vasticrureus muscle, inaugurated by single knee-jerk. Rate of clonus, 11·5 jerks per second. Time, below, 13·5 beats per second.

this experiment was 11·5 complete contractions per second. Other records of patellar clonus were 11·2, 12·6 and 11·5 complete contractions per second. In still other animals the rate was higher, 17·2, 17·1, 17·3 and 16·4 complete contractions per second. There seemed to be, therefore, two rather definite rates, one about twelve complete beats and

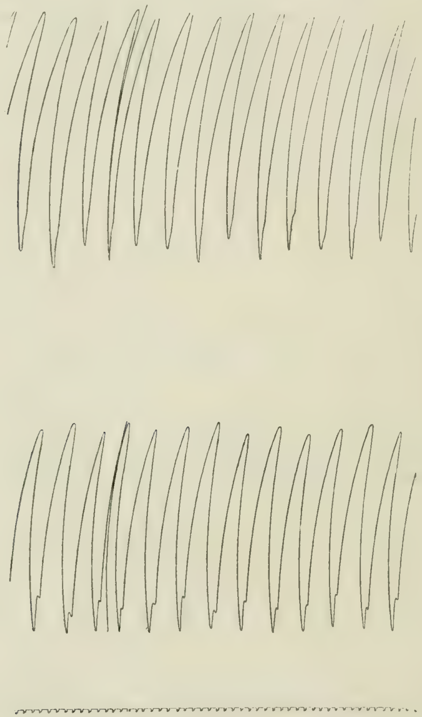

FIG. 10.—Reflex "walking" in a decerebrate cat; two vasticrurei muscle preparations. Alternate contraction of the two muscles. Rate, 3·5 steps per second. (For full description, see text.)

another about seventeen complete beats per second. The rate was always nearly constant in the same animal.

One case of patellar clonus was studied in man, using the same apparatus. The case was one of a hemiplegia of some years' duration with marked spasticity of the left leg, increased knee-jerks, patellar

clonus, ankle clonus and a positive Babinski reflex. The rate of the patellar clonus in man was between 8·8 and 9·7 complete beats per second.

By way of comparison with the clonus rate some records were obtained of the "walking reflex," an alternate contraction and relaxation of the vasticrurei, and fig. 10 shows a record of the two extensor muscles during the walking reflex. The rate of this walking at the time this record was taken was 3·5 steps per second, but great variations in the rate were observed. At one time it was recorded that the rate was 1·7 steps per second and at another 2·7. It will be seen, therefore,

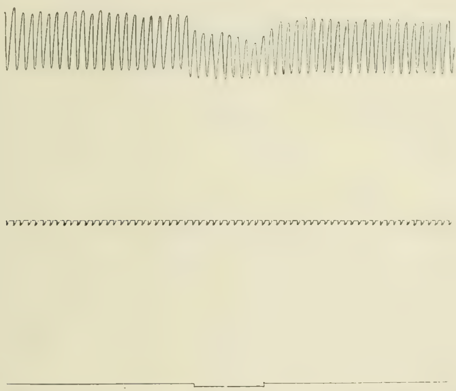

FIG. 11.—Patellar clonus in a decerebrate cat; vasticrureus muscle. Tonus inhibited by ipsilateral peroneal stimulation, indicated by signal, lowest line. Time, middle line, 13·5 beats per second.

that the walking reflex is quite different from the patellar clonus. The patellar clonus is very constant in the same animal, although it may be of different rates in different animals. The walking reflex, on the other hand, varies in rate from time to time in the same animal.

A double clonus was never successfully recorded in any of the experiments, although it was observed a number of times. The contractions were synchronous, not alternating as in walking. Clonus was not always obtained in decerebrate cats, but appeared at unsuspected times, often when our apparatus was not adjusted for recording owing to mechanical difficulties. We were never able to obtain clonus by any special form of decerebration and it was always observed incidentally while working on the form of the knee-jerk.

Variations in the form of the clonus were observed during changes in the amount of clonus exactly corresponding to variations in the form of the knee-jerk during changes of the tonus. Fig. 11 is a tracing of an experiment in which the tonus was decreased by an ipsilateral peroneal stimulation during the recording of a clonus. The record shows the drop in tone and also that the clonus changed slightly in character. The rate remained the same, but the clonus became irregular with variations in the size of the jerk. When the inhibition of the tonus was released the clonus gradually "climbed" and sought the same

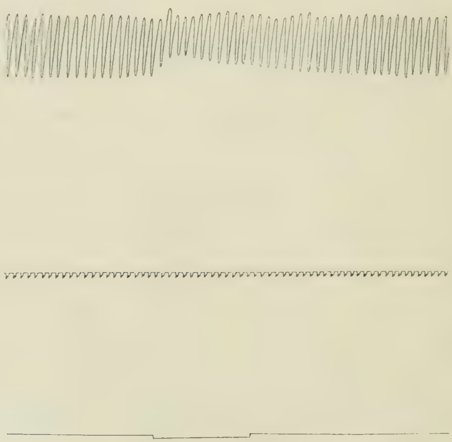

FIG. 12.—Patellar clonus in a decerebrate cat; vasticrureus muscle. Tonus augmented by a weak contralateral peroneal stimulation, indicated by signal, lowest line. Time, middle line, 13·5 beats per second.

level (i.e., tone) as before inhibition. On the other hand, when the tone was increased by contralateral peroneal stimulation the same phenomena were observed (fig. 12). There was a decrease in the amplitude with a slight irregularity. If the tonus was decreased or increased markedly the clonus would at once stop and it was observed that only between certain degrees of tonus (*i.e.*, certain levels on the drum) could clonus be obtained, and any tonus above this level or below this level was sufficient to stop the clonus.

IV.—Discussion.

There are two distinct forms of the knee-jerk noted in these experiments. One is the simple jerk with the contraction and relaxation of about the same degree, as seen in the spinal animals. The other is the decerebrate knee-jerk in which in addition to the twitch there is a plastic element which holds the muscle at a new degree of tonic level after the jerk. This second element in the decerebrate knee-jerk was shown by Sherrington [7] in his experiments on the shortening and lengthening reactions. He showed that the spastic muscle in the decerebrate cat was capable of being "set" at various lengths and would remain so for an indefinite period. These two elements in spastic muscles in decerebrate animals have been noted by other investigators. Langelaan [3] proposed the terms "contractile tonus" and "spastic or autonomic tonus," and clearly showed them in his experiments on animals. He concludes "that the contraction following the tap on the tendon is of a twofold nature, viz., a twitch upon which is superposed a tonic contraction."

Plasticity as a part of spasticity has not, so far as I am aware, been adequately demonstrated in man except for Wertheim Salomonson's paper. He obtained, in a case of cerebral diplegia, curves of the knee-jerk exactly corresponding to the spastic curves seen in the cat (fig. 2.) He noted that "the muscle does not entirely relax, but a state of hardening of the muscle substances persists for a longer or shorter time." From the illustrations he uses, the "hardening," I think, is the same as the plastic tonus described by Langelaan and others. The twitch is followed by a continued contraction as in the experiments on animals. I have, however, noted plasticity in some of the severe cases of hemiplegia and in one case of diplegia, but have never recorded this graphically. The so-called "lead pipe" rigidity is, I think, an example of plasticity combined with spasticity in man. Nevertheless it is certainly not the usual finding in a spastic muscle in man, as the most noticeable phenomenon is the great increase in the amplitude of the responses to the patellar tap. Thus the laboratory finding, where plasticity is the usual companion of spasticity, is not always easily demonstrated in the clinic.

In the laboratory, however, we are observing animals whose nervous system is far less integrated than the nervous system of man. The activity of the spinal animal where reflexes may be demonstrated in almost pure form is much greater than the "spinal man." It must be recognized, nevertheless, that many of the reflexes such as the scratch

reflex and the extensor thrust, so clearly demonstrated by Sherrington in animals, are present in man, although they may be so weak or changed that they are not easily found. The work of Head and Riddoch [2] on certain reflexes in cases of complete spinal cord lesions without infections shows that many more reflexes may be seen in the isolated human spinal cord than was previously supposed. Riddoch [5] found that the usual knee-jerk in his cases of complete section of the spinal cord was a simple twitch, presumably the same as is shown to be the case in spinal animals (fig. 1). In one of his cases, however, where there was not a complete transection of the cord he found after the twitch of the knee-jerk "the leg fell away more slowly and the relaxation of the tendon was more deliberate." He thought in this case there was some "plastic tonus," as well as "contractile tonus," to use Langelaan's term. Walshe [10] has also successfully searched in men for reflex actions that Sherrington had demonstrated in the laboratory.

It is, perhaps, not remarkable that the plastic element in spasticity has not been observed more often in man. It must be remembered that one seldom has a chance of seeing in man an isolated reflex group such as is artificially produced in animals. Also, the extraneous factors which I mentioned in the early part of this paper are so profound that one cannot observe a pure reflex contraction without the action of other muscles on the same joint or inhibition or augmentation of the reflex by voluntary control. It was observed in these experiments that it was not possible to obtain a knee-jerk in an absolutely toneless muscle. In the spinal animal the vasticrureus muscle was not entirely toneless, for we were able to inhibit some tonus by ipsilateral peroneal stimulation. In other words, we were able to decrease its tonus and finally a point was reached where no reflex was obtained. This, I think, demonstrates that tonus is not entirely dependent on control from centres in the mid-brain, for when the spinal cord is entirely cut across we have severed all connection with such centres. Such a spinal animal has very little tone and we speak of the muscles as flaccid, but nevertheless there is some tonus left. This point, too, has been demonstrated in man by reflexes obtained after complete transverse lesions of the spinal cord. Such reflexes I believe are not possible without some tonus.

It was noticed that the spastic vasticrureus muscle when "set," well lengthened, and then stimulated by blows on the patellar tendon, tended to shorten at each jerk until a certain level was reached on the recording

drum, and then tended to remain at that level. In other words, the muscle found a length between its high and low tonus length, at which point there was a marked tendency to remain. This level was also shown in the experiments where the tonus was decreased or increased by ipsilateral or contralateral peroneal stimulation ; after such stimulation the muscle took what I have called a "neutral tension." Two factors seem to control this tension, one the reflex tonus tending to increase the tension of the muscle and the other the weight of the limb tending to pull it down and thus lengthen the muscle. When the reflex tonus was very great the muscle was shortened as far as the knee-jerk would allow, the limb being completely extended and the weight of the limb being completely overcome. The point of interest is that at a certain level of tonus, clonus was most apt to appear. It is at the neutral tension of the muscle somewhere between its high and low tonic level that clonus is most apt to appear. In other words a muscle at this tonic tension is in its best position to maintain a continued series of contractions. Clonus was obtained mainly in the spastic muscles at their neutral tension. If a spastic vasticrureus was set at a low level of tonus and the patellar tendon tapped, the plasticity after the jerk brought the muscle to the most favourable level before clonus would appear. If the tonus during a clonus was increased or decreased by contralateral or ipsilateral peroneal stimulation and passed beyond the narrow limits of its neutral tension the clonus either became irregular or disappeared. Langelaan has noted that "a clonus is composed of a series of twitches superposed upon a tonic shortening." This, I think, is correct in a measure, but the tonic shortening (or increased tension) must be a definite one, for any increase or decrease in the tension of the muscle beyond the narrow limit of its neutral tension is enough to cause the clonus to stop. Wertheim Salomonson found clonic after-contractions (pseudoclonus) in many of his cases and in others stated that "the tonus is not quite high enough to permit of a clonus." He showed no records of a true clonus.

It would seem then that a true clonus is a series of continuous reflex twitches dependent upon a more or less fixed tone of the muscle. Clonus is possible only over a slight range of tonus, for there is a tonic tension of a muscle, a "neutral tension," that is, the tone "of choice," for a series of continued self-sustaining twitches. If the plastic element holds the muscle at this length, then the contractile force will produce the jerk, and a clonus will result.

The clonus, as we have noted, is a regular series of twitches. If the

tone is raised or lowered, the clonus becomes irregular in both amplitude and rate. Thus we have in our plastic tonus the mechanism by which such a continued muscular effort as the clonus is controlled, so that it continues regularly and smoothly. The tonic level is so regulated by plasticity that the muscle is set at a correct height, so that the falling limb is just enough stimulus to provoke the next twitch. This seems to me one of the functions of plasticity, and its value in animal economy cannot be doubted.

Conclusions.

Experiments on cats, both spinal and decerebrate, with the anterior crural-vasticrureus nerve-muscle preparation isolated in situ, and the movement of the knee-joint recorded graphically, gave the following results:—

(1) In the records from the spinal animal the knee-jerk is a single twitch, the contraction and relaxation of the extensor muscle group being almost parallel. After the jerk there may be another jerk ("rebound") or a short series of jerks ("pseudoclonus"), but the length of the muscle after each jerk is the same as before the experiment.

(2) In the records of the decerebrate animal the knee-jerk is a single twitch followed by a continued contraction, the muscle assuming a new and shorter length after the jerk. Thus, there are two elements in the phenomenon, the twitch or jerk and the continued contraction. The first may be termed the "contractile element," and the second the "plastic element," after Langelaan.

(3) The plasticity is an indicator of muscle tonus. When the tonus is varied by artificial means, the amount of plasticity varies also. When the tonus is lowered by an ipsilateral peripheral nerve stimulation, the plastic element may drop out of the knee-jerk record, and the jerk takes the form seen in the spinal animal.

(4) Some tonus can be demonstrated in the flaccid vasticrureus muscle of the spinal animal, for it may be reduced by ipsilateral peripheral nerve stimulation, showing that the distal portion of the completely severed spinal cord in a cat is capable of maintaining some tonus in the muscles of the lower limbs.

(5) Many of the knee-jerks in both spinal and decerebrate animals may show pseudoclonus; but it is most often found in decerebrate preparations, showing little tonus.

(6) A true clonus may be obtained in various ways. If the tonus of a spinal animal is raised by contralateral peripheral nerve stimula-

tion, a clonus may result. Decerebrate animals with moderate tonus most often exhibit clonus. The degree of tonus of the muscle at the time of clonus is termed its "neutral tension." This tension is a neutral point between the reflex tonus, tending to increase the tension of the muscle and weight of the limb tending to lengthen it. This tension, and this tension only, is the most favourable point for the maintenance of a clonus.

(7) A true clonus is characterized by a continued series of self-sustained reflex twitches, regular in rate and amplitude, markedly altered or stopped by increasing or decreasing the tonus.

(8) The rate of a clonus in the vasticrureus muscle group of a cat was found to vary, most of the records falling into two groups—a rate about 12 per second and another about 17 per second. Patellar clonus in man was found to be 8·8 to 9·7 per second.

(9) The rate of "walking" varied from 1·75 to 3·5 steps per second.

BIBLIOGRAPHY.

[1] Bowditch and Warren. *Journ. Physiol.*, 1890, vol. xi, p. 25.
[2] Head and Riddoch. *Brain*, 1917, vol. xl, p. 188.
[3] Langelaan. *Brain*, 1915, vol. xxxviii, p. 235.
[4] Lombard. *Journ. Physiol.*, 1889, vol. x, p. 122.
[5] Riddoch. *Brain*, 1917, vol. xl, p. 264.
[6] Sherrington. *Journ. Physiol.*, 1909, vol. xxxviii, p. 375.
[7] *Idem.* *Quart. Journ. Exp. Physiol.*, 1909, vol. ii, p. 109.
[8] *Idem.* "Integrative Action of the Nervous System," 1906.
[9] *Idem.* *Brain*, 1915, vol. xxxviii, p. 210.
[10] Walshe. *Brain*, 1914-15, vol. xxxvii, p. 269.
[11] Weir, Mitchell and Lewis. *Trans. Assoc. Amer. Phys.*, 1886, vol. i, p. 2.
[12] Wertheim Salomonson. *Proc. Roy. Acad.*, Amsterdam, 1915, vol. xvii, p. 885.

ALTERATIONS OF THE GOLGI APPARATUS IN NERVE CELLS.[1]

BY WILDER G. PENFIELD, M.A., M.D.

Retispersion.

An intracellular reticulum was first described by Golgi [10] in 1898, in the cells of the central nervous system. This observation has been greatly extended, first by his pupils in Pavia, and subsequently by a large field of workers, until at the present time the structure has been described in the cytoplasm of practically all types of cell throughout the animal kingdom [24, 6, 7, 21]. The structure has received a variety of names, such as the internal reticular apparatus of Golgi [10], the "Binnennetz" of Kopsch [15], the canalicular apparatus (Bensley [1]), &c.

This study is concerned only with the apparatus as seen in nerve cells, where it appears in its most highly differentiated form and presents normally great variation in appearance. There have been numerous theories as to its function in the cell. But before a study of this problem can be logically undertaken, an inquiry must be made into the alterations of this structure in varying nerve cell conditions. Such an inquiry is the purpose of this paper.

The Golgi apparatus presents normally, in the great majority of cases, a complete attenuated reticulum with many varicosities or lacunæ (figs. 1 and 2). These varicosities may be homogeneous and black, as though full of some substance in which the silver is deposited, or they may frequently appear empty of such substance, and only the surface or boundary of the space take on the silver. The structure is confined to the cytoplasm, never encroaching on the nucleus or the periphery of the cell. Threadlike prolongations may pass outward into the dendrites, but I have never seen them in the axone process, nor have I found any such description in the literature.

Variations from the complete reticulum are most frequently seen in

[1] This research forms part of the work done for the degree of B.Sc., in the Physiological Laboratory, Oxford University. Expenses were defrayed partially by the Christopher Welsh Scholarship Fund.

the spinal ganglion cells. The whole structure appears rarely in one half of the cytoplasm only. It may also, occasionally, be fragmented to a greater or less degree. It may be hypertrophied or meagre, but under normal conditions the general pattern is surprisingly constant for each type of nerve cell.

A Zeiss objective, homog. immers. 1·5 mm., and a 6 compensating eye-piece (magnification 1,000) were used for all drawings, which were made with the aid of an Abbé camera lucida, Zeiss. All sections were prepared by the Cajal uranium-formol-silver method without counterstain. All material was obtained from cats.

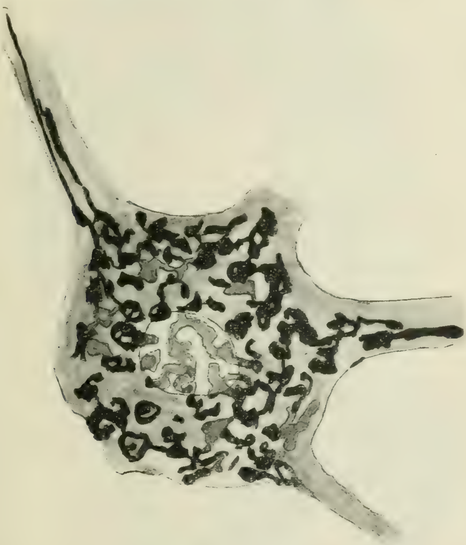

FIG. 1.—Normal cell in anterior horn of spinal cord. That part of the Golgi apparatus which is grey instead of black was drawn with the focus at a deeper level, below the nucleus.

THE LITERATURE OF ALTERATIONS IN THE GOLGI APPARATUS.

We have to thank the brilliant researches of S. Ramón y Cajal and his pupils for the larger part of our knowledge of this subject.

Autolysis.—Autolytic changes in the Golgi reticulum are rapid, beginning in man, according to Cajal, two hours after death. At twenty-four hours the reticulum has become granular, and in some cells only fine fragments will be found to remain.

In the cells of transplanted ganglia, the apparatus shows similar changes [23, 24].

Trauma.—In 1914, when studying the changes following traumatic inflammation, Cajal [23] concluded that the reticulum is more resistant

FIG. 2.—Normal cells in spinal ganglion. About both nerve cells may be seen the subcapsular cells, whose Golgi apparatuses are located at one pole of the respective nuclei.

to pathological agents than are the neurofibrils. But he found in the nerve cells of the cord and the pyramidal cells of the cortex that direct trauma causes fragmentation of the reticulum and its displace-

ment to the cell periphery and into the bases of the dendrites. The cell undergoes a process similar to chromatolysis, and as the nucleus approaches the periphery fragments of the Golgi apparatus frequently gather near it. The reticulum may disappear altogether. Therefore, in chromatolysis subsequent to direct injury, the Golgi apparatus and Nissl granulations behave somewhat similarly, both moving out to the periphery of the swollen cell and sometimes disappearing. In later stages, six to twelve days after the wound, he found on its border nerve cells with frankly hypertrophied Golgi apparatus.

Faradism.—Legendre [16] stimulated the posterior root of a lumbar spinal ganglion. He found that, as a result, the Golgi net in each cell was displaced to the periphery and became somewhat deformed. It is not stated what precautions were used to prevent spread of the electrical current along the root to the ganglion direct. Holmgren [12, 13] also tried the effect of electrical stimulation on nerve cells and found that, accompanying chromatolysis of the Nissl bodies, there resulted (*a*) "Kanalizirung," an emptying of the canals, and (*b*) diminution in size of the trophospongium, a system of canals having extracellular connections, which he maintains [14] is identical with the Golgi apparatus.[1]

Lesion of Axone.—Marcora [17, 18] found that, subsequent to section or tearing out of the hypoglossal nerve in a rabbit, there developed a fragmentation and displacement of the intracellular apparatus toward the periphery, which was well marked on the fourth day and reached its height between the fifteenth and eighteenth days. He also noted hypertrophy of the Golgi apparatus after the immediate effect of section had passed.

Cajal [24] described lobulation and fragmentation of the motor cells of the spinal cord after section of the corresponding nerves. He stated that this change accompanied chromatolysis, and, as the nucleus approached the cell periphery, the remains of the broken reticulum often gathered about it, or in the bases of the dendrites. He found no change in the Golgi apparatus of the cells of Clarke's column after section of their axones and, similarly, after section of the sciatic, he reported the spinal ganglia of that side unchanged. He concluded that section of the peripheral extension of a sensory neurone is not a

[1] The question of the identity of these two structures is discussed in a paper to appear shortly [22]. For an excellent summary of the literature on this subject, see Duesberg [6, 7].

sufficient cause to produce noticeable changes in the Golgi apparatus of that cell.[1]

It is of interest that Cajal [25] showed changes of the internal reticulum in the cells of the sheath of Schwann on the peripheral stump of the nerve after section. The reticulum became increased in quantity, occupied almost the whole width of the sheath, and extended along the fibre in either direction from the nucleus in parallel lines.

Nissl Granulations—Chromatolysis.

Inasmuch as many of the changes reported below in the Golgi apparatus are associated with somewhat similar changes in the Nissl granulations (chromaphil substance), it may be of interest to review briefly our knowledge of the latter and the inter-relation of the two substances.

The most delicate morphological index of the functional activity of a nerve cell is generally considered to be the appearance of the chromaphil substance in the cytoplasm, the so-called Nissl granulations.

Alterations in the appearance of this substance caused by electrical stimulation (Nissl, Vas, Lambert, Mann, Lugaro, Pugnat, and others) consist in a progressive diminution associated, in some cases, with displacement of the granules toward the periphery of the cell. Faradic stimulation creates abnormal conditions, however, which are probably not reproduced in the physiological activity of the nerve cell.

The results obtained by a long series of investigators (Van Gehuchten [30], p. 300) indicate that, following normal cell activity, there is a diminution in the amount of Nissl substance, which grows more marked with the onset of fatigue.

After section of the nerve, there may result, as first pointed out by Nissl [20], a change in the cells, the most prominent features of which are: disappearance of the Nissl substance, a considerable increase in the size of the cell, and displacement of the nucleus toward the cell periphery. This phenomenon was called by Marinesco chromatolysis, inasmuch as the alteration in the Nissl substance seemed to be its most striking characteristic. The first signs of change may appear as early as forty hours after the section. (Van Gehuchten [30], p. 305.) The Nissl

[1] Page 69 of reprint from Reference 24. "Debemos, pues, concluir, que la sección de la rama periférica de la neurona sensitiva no es causa suficiente para provocar perturbaciones marcadas en el aparato de Golgi de esta célula."

granules begin to disappear at the centre of the cell, and are visible last about the periphery and sometimes close to the nucleus. Following this phase of "dissolution," which reaches its height after fifteen to twenty days, there ensues a phase of "reformation" which progresses rather more slowly until the cell is again normal in appearance. A certain proportion of cells never recover, but go on to atrophy. The duration and degree of chromatolysis depend on the type of injury to the axone. If the nerve be torn out, the reaction is more severe and certain than that which follows section, ligature, or pressure of the nerve.

In this paper we are most interested in the cells of the spinal cord and ganglia. The motor cells of the anterior horns of the cord appear to offer considerable resistance to section of the axone. At times there is no resultant chromatolysis (Van Gehuchten [29]). After a lesion of the peripheral neurone-outgrowth of a spinal ganglion cell, chromatolysis is rapid, but section of the central fibres gives rise to no evident reaction. Section of the spinal cord produces in the cells of Clarke's column, located below, an energetic and typical response which is eventually followed by complete atrophy of at least the large cells in the column. (Sherrington and Laslett [28].)

The Golgi Apparatus and Nissl Granulations.

In 1910 Legendre [16] proposed that these two structures were identical. He called attention to the fact that, in general, their distribution was the same, both being situated in the cytoplasm, separated from the cell periphery by a clear space and avoiding the axone hillock. When the Nissl granulations were small in size, the Golgi net was composed of small granules and fine filaments. After electrical stimulation of the nerve, he noted similar alterations of the two structures. In transplanted ganglia, he observed the disappearance of the net and granulations at about the same rate.

This close association of the two structures is of interest, but that Legendre was mistaken as to their identity has been amply proven by the observations of other workers. Collin and Lucien [4] impregnated the Golgi apparatus and stained the Nissl granulations in the same sections. They found a certain number of cells where the one was perinuclear and the other peripheral. Marcora [17] stained the two structures simultaneously and demonstrated the Nissl bodies lodged in the mesh of the Golgi net; and in embryonic tissue [19] he showed that the two were separated by a clear unstained space. Cowdry [5] demonstrated the independence of Nissl bodies, "canalicular apparatus,"

and also mitochondria and neurofibrils in the same cell, by the Bensley method.

Cajal [24] agrees to the similarity of distribution of the two protoplasmic structures with regard to the peripheral freedom of the cell from both and the resemblance of a fragmented reticulum to the Nissl granules. But he calls attention to the fact that, whereas the Nissl bodies are found in the nerve cells only, the reticulum of Golgi is found generally in cells of all descriptions. He reports that his preparations, uranium nitrate-silver impregnation followed by staining of the chromophil bodies with aniline dyes, show constantly a clear space surrounding the Golgi reticulum and separating it from the Nissl substance. He states that this clear space is certainly occupied by neurofibrils.

Technique.

In the following experiments the Golgi apparatus was demonstrated by Cajal's uranium-formol-silver method [24],[1] with several minor modifications, as follows:—

The tissue was removed from the body immediately after death. Blocks of the cord must be very thin, not over 1 to 2 mm. After an hour in the fixative, blocks may be removed and cut down more easily to the desired thickness.

(1) Fixation for twelve hours. Shake from time to time. Uranium nitrate, 1 grm.; commercial formol (40 per cent.), 20 c.c.; distilled water, 80 c.c. The formol should be neutral. It is sufficient to keep chalk in the bottom of the container. For the cord and spinal ganglia 20 c.c. of formol gives more consistent results than 15 c.c. as suggested by Cajal. It is best to make up solutions of formol and of uranium nitrate separately, of twice the required strength, and mix in equal parts, just before use.

(2) Impregnation for forty-eight hours. After washing in distilled water for a few seconds only, immerse in 1·5 per cent. silver nitrate. Shake from time to time.

(3) Reduction for twelve hours. (First wash in distilled water.) Hydroquinone, 2 grm.; formol (commercial), 20 c.c.; sodium sulphite, 0·5 to 1·5 grm.; distilled water, 80 c.c. Sufficient sodium sulphite should be added to turn the solution quickly to a coffee colour.

(4) Dehydrate very rapidly in alcohol. Pass through xylol to wax.

[1] An excellent summary of this method has been published in English by Carleton [2].

From 90 per cent. alcohol until the tissue is embedded should not occupy more than five hours. If this stage is not carried out rapidly it may be found that all silver will have disappeared from a peripheral zone of cells in the spinal ganglion sections.

The fixation is apparently not sufficient to prevent the subsequent solution of some or all of the constituents of the Golgi apparatus in strong alcohol (Gatenby [9]), if exposed to it for too long a time. Toning of the sections with gold chloride adds nothing.

In two experiments I obtained the best results and perfect fixation of the cord by subarachnoid injection of the fixative before death, as follows: The animal is anæsthetized and a laminectomy performed in the lower lumbar region, exposing the membranes. A medium-sized lumbar puncture needle is then passed into the subarachnoid space a short way upward beside the cord and the fixative allowed to flow in under a gravity pressure of 75 cm. The heart stops after about a minute of the injection, which is continued for twenty hours. At the end of this time the cord is removed, blocks cut and dropped directly into the silver nitrate bath. Likewise, sections from tissue which has been fixed in this way and left a few days in 95 per cent. alcohol give the most perfect result when stained by Nissl's method. Subarachnoid injection does not fix the spinal ganglia properly, if at all.

This method, in general, if adapted to the type of cells one wishes to study, gives very complete and fairly consistent impregnations of the Golgi apparatus. In my experience, strength of fixative and period of fixation are the most important elements in the adjustment of the method to any particular tissue. In the sections prepared for this study there was also impregnation, at times, of the mitochondria in the nerve cells, and the Golgi apparatus in both the neuroglia cells of the cord and the intracapsular cells (Holmgren's "trophocytes") of the spinal ganglia. In the ganglion cells an intra-nucleolar body (*see* Carleton, [3]) was frequently impregnated.

When counterstain was desired, Unna's polychrome methylene blue was employed, demonstrating Nissl granulations, nuclear structures, &c.

For the purpose of this research, cats were used, under two to three months of age as a rule. In the few experiments where older cats were used, the impregnation of the Golgi apparatus (contrary to the experience of Cajal) was equally satisfactory. The frequent presence of well-stained pigment granules, however, in the material from the older animals, added another cytoplasmic constituent.

Results of Experiments.

To reconsider briefly some of the foregoing paragraphs: As a result of section of the axone, changes in the Golgi apparatus have been described in the motor cells of the cord and bulb, but none in the cells of the spinal ganglia or Clarke's column. The nature of the reported change is progressive fragmentation and lobulation of the reticulum with some peripheral displacement. Chromatolysis appears in the cells of the anterior horn and Clarke's column after axone section, and

Fig. 3.—Retispersion in anterior horn cell of left side of spinal cord. Level of 7th lumbar segment. Seven days after section of left sciatic nerve.

in spinal ganglion cells after section of the peripheral extension of the axone only. Cutting of the central extension fails to cause the reaction. My observations give no cause to modify these conclusions, at least in so far as the phenomenon of chromatolysis is concerned.

Positive results.—The work reported here indicates that the Golgi apparatus in all types of neurones studied, afferent as well as efferent, responds to an axone lesion in a consistently specific manner.

The *motor cells* of the cord were studied after simple section of the sciatic nerve. There was no possibility of trauma as the section was made at the level of the great trochanter, a distance of about 5 cm. below the seventh lumbar ganglion when measured along the nerve. At the end of seven days, after the cutting of the nerve on one side, the reticulum had begun to leave a clear zone about the nucleus of the motor cells on that side. This could be made out in 22 per cent. of the cells counted. No such condition was found in the examination of an equal number of cord neurones on the opposite side, where the nerve was intact. Aside from this peripheral displacement, there was very little change in the appearance of the apparatus (fig. 3). After sixteen days the change had become more general, the clear perinuclear zone was wider and now appeared in 50 per cent. of the homolateral motor cells, the heterolateral side being 100 per cent. normal. There was now a moderate amount of fragmentation in some cells, while in others the reticular structure remained unbroken. The neighbourhood of the axone base becomes free of all Gogli apparatus early in the process.

It seems desirable to call this process by a distinctive name, *retispersion* (from *rete*, a net, and *spargo*, to scatter), especially since it may appear independently of chromatolysis as will be shown below. After this stage of dispersion of the Golgi apparatus to the cell periphery, there may succeed a stage of dissolution, when the reticulum becomes progressively fragmented. Thus *retispersion* may be followed by *retisolution.* It seems evident, however, that the amount of retisolution varies with the different types of neurone.

The cells of *Clarke's column* respond in a most energetic fashion. In Experiment 14, the spinal cord of the cat had been completely severed four days previously. The section was at the level of the eleventh dorsal spine. In the lower lumbar region, the cells which correspond to Clarke's column higher in the cord showed a striking change. The Golgi reticulum was close against the periphery of the cell except where some remained about the nucleus, which also had become eccentric. In spite of its extreme dislocation, however, the network of the apparatus was complete for the most part (fig. 5). Retisolution had begun only in a few cells (fig. 6). Nissl granulations were absent.

The *spinal ganglion* cells present normally such a rich variety of types of Golgi apparatus that considerable care must be taken to control all conclusions. Four days after section of the left sciatic, retispersion had begun in the spinal ganglion cells of the same side. The Golgi apparatus had moved outward toward the periphery, leaving a clear

space about the nucleus in 48 per cent. of these cells, whereas in the same ganglion on the opposite side the apparatus was more or less peripheral in only 16 per cent., which variation is about the average for

FIG. 5.—Retispersion in neurone of Clarke's column in lumbar spinal cord. Four days after section of spinal cord at level of 11th thoracic spine.

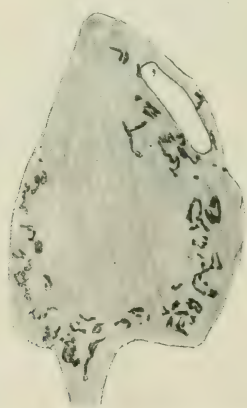

FIG. 6.—Same as fig. 5, showing considerable retisolution as well.

normal ganglion cells. The difference,[1] of course, is more especially one of degree of displacement, which cannot be estimated in figures. Nissl stains showed no evidence of chromatolysis as yet.

[1] If sections are cut thin, at about 6 μ, retispersion is less apt to be overlooked than when the sections are thick.

Seven days after the sciatic was cut, the peripheral dislocation of the reticulum reached its height, affecting 80 per cent. of the homolateral cells as compared with a count which was again 16 per cent. for the

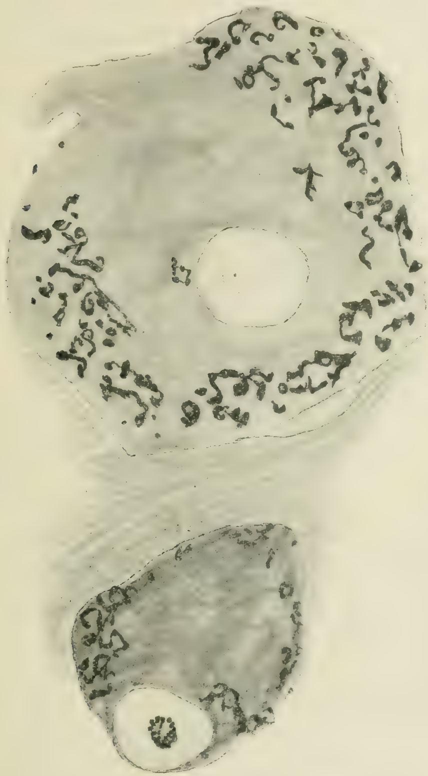

FIG. 4.—Retispersion in neurones of 7th lumbar ganglion, left side, showing the clear space about base of axone. Seven days after section of left sciatic nerve. Same experiment as that of fig. 3.

opposite side (fig. 4). Now, however, fragmentation of the apparatus had begun somewhat and also chromatolysis was present in many of the cells. On the sixteenth day chromatolysis was at its height, but retispersion seemed to have passed its apex. Although in certain cells

retisolution was more extreme than in any seen previously, there was a larger proportion of cells with a complete Golgi apparatus in its normal position than on the seventh day. In the larger cell (fig. 4) the absence of reticulum from the neighbourhood of the axone is striking. In general, that part of the cell which becomes free from all Golgi apparatus has the shape of a pear whose stem corresponds to the axone (see also fig. 3).

Interest naturally turns next to an inquiry as to whether cutting of the central extension of the axone of the spinal ganglion cell is followed by retispersion. According to Van Gehuchten, as has already been stated, this cutting of the central fibres is, curiously enough, not followed by chromatolysis ([30], p. 316). The posterior nerve roots proximal to the sacral and lower lumbar spinal ganglia were cut. Seventeen days later, retispersion was present, although not extreme. Double staining by the Cajal and Nissl methods showed the Nissl bodies for the most part normal in amount and location, although in a few cells they could be stained only at the cell periphery in the mesh of the Golgi apparatus.

The attempt was next made to produce retispersion in those sensory cells some of whose fibres pass upward through the cord without interruption. The spinal cord was cut and the ganglia lying below examined; but no conclusive evidence of a resultant change could be made out two, four, six, or twelve days after section or semisection of the cord. It must be borne in mind, however, that only a part of the central neurone-outgrowth could have been thus severed. Other divisions of the neurone end below the site of section.

Negative results.—Four and twelve days after section of the cord above, no change was found in the Golgi apparatus of the anterior horn cells. Seven days after an aseptic decerebration performed on a cat by H. C. Bazett (to whose kindness I am indebted for the material), no changes could be found in the cord motor cells. One must conclude that the alterations in the activity of the reflex arc following cord section or decerebration, are not reflected by any detectable change of the Golgi apparatus in the motor cells.

Local tetanus was produced in one hind limb of cats by opening the sheath of the common peroneal nerve and placing in and about it 30 to 60 mgm. of powdered tetanus toxin. Rigid tetanic extension of the limb followed in about three days. Six, twelve, and seventeen days later no definite change could be observed in the cord or ganglion cells corresponding to the tetanized limb.

The injection of a lethal dose of strychnine (0·83 mgrm.), once repeated, in a decapitated preparation (Sherrington [27], pp. 137-141) produced recurring tonic and clonic spasms over a period of four hours. Subsequent examination of the Golgi apparatus in the spinal cord cells revealed no changes.

If, therefore, it is possible to produce a condition of fatigue in the cells by tetanus or strychnine convulsions, at least there is no associated change apparent in the Golgi apparatus.

Summary of Results.

Decerebration and high section of the cord are without effect on the Golgi apparatus of the anterior horn cells. Following tetanus, there were no changes in the cord or ganglion cells. Administration of strychnine, causing repeated convulsions, likewise gave a negative result. The increased muscular tone of decerebrate rigidity and the muscular flaccidity following cord section must be associated with considerable variation in the activity of the motor cells. Likewise, the constant muscular contraction of tetanus, which is sufficient to cause actual muscular wasting, indicates greatly increased activity on the part of the motor cells. Nevertheless, under the above conditions, I have not been able to discover any change in the Golgi apparatus of the neurones in the spinal cord or ganglia.

Section of the axone is followed by retispersion, a specific alteration of the Golgi reticular apparatus in nerve cells. The complete reaction is made up of three stages: (1) Displacement of the unbroken apparatus to the periphery of the cell and away from the axone-hillock—retispersion (figs. 3 and 5). (2) Dissolution of the reticulum—retisolution (fig. 6); frequently there is very little evidence of retisolution. (3) Reconstruction.

In general, chromatolysis and retispersion are associated, but they may appear independently. Retispersion has been the first to appear in the experiments reported here. In a particular cell, the reticulum may be peripheral or even absent, while the nucleus remains central and the chromophil substance appears normal.

Retispersion appears early in Clarke's column, being far advanced on the fourth day after cord section. The motor cells seem to be more resistant to alteration. The Golgi apparatus appeared at the periphery of a small proportion of these cells on the seventh day and this became quite general on the sixteenth day after cutting the sciatic, but fragmentation was infrequent.

In the spinal ganglia, after section of the peripheral neurone-outgrowth, the fourth day saw the beginning and the seventh day the height of retispersion, with the appearance, also, of considerable retisolution. The cutting of the central division of the axone-outgrowth, the posterior nerve root, induced retispersion in the corresponding ganglia. But section of the cord failed to produce any change in the ganglia below, probably due to the fact that all of the central fibres could not have been thus severed.

To sum up briefly: In the above experiments, no changes were observed in the Golgi apparatus, following variations in nerve cell activity. The apparatus showed no detectable alteration after the removal of the influence of *connecting* neurones, but it exhibited a delicate and specific response, retispersion, to a lesion of its axone-outgrowth, whether central or peripheral. Retispersion is an added feature of the cellular reaction to axone interruption, usually called chromatolysis. It is ordinarily the first and in some cases the only manifestation of this cellular reaction.

In conclusion, it is a pleasure to thank Professor Sherrington for his interest and helpful criticism; also, Mr. H. M. Carleton and Dr. J. B. Gatenby for many useful suggestions.

BIBLIOGRAPHY OF CITED LITERATURE.

[1] Bensley, R. R. "On the Nature of the Canalicular Apparatus of Animal Cells," *Biol. Bulletin*, Woods Hole, Mass., 1910, vol. xix, pp. 179-194.
[2] Carleton, H. M. "Note on Cajal's Formalin-silver Nitrate Impregnation Method for the Golgi Apparatus," *Journ. Roy. Micr. Soc.*, 1919, pp. 321-328.
[3] *Idem.* "Observations on an Intranucleolar Body in Columnar Epithelium Cells of the Intestine." *Quart. Journ. Micr. Sci.*, 1920, vol. lxiv, pp. 329-343.
[4] Collin, R., and Lucien, M. "Réseau interne de Golgi dans les cellules nerveuses des Mammifères," *Compt. rend. Assoc. des Anat.*, 1909, t. xi, pp. 238-245.
[5] Cowdry, E. V. "The Relations of Mitochondria and other Cytoplasmic Constituents in Spinal Ganglion Cells of the Pigeon," *Internat. Monatschr. f. Anat. u. Physiol.*, 1913, vol. xxix, pp. 473-498.
[6] Duesberg, J. "Plastosomen, 'Apparato reticolare interno' und Chromidialapparat," *Ergebnisse der Anat. u. Entwickelungsgeschichte*, 1912, Bd. xx, S. 567-916.
[7] *Idem.* "Trophospongien und Golgischer Binnenapparat," *Anat. Anz.*, 1914, Bd. xlvi, Ergänzungsheft, S. 11-80.
[8] Gatenby, J. "The Cytoplasmic Inclusions of the Germ Cells. Part V. The Gametogenesis and Early Development of the *Limnea Stagnalis* (L.), with Special Reference to the Golgi Apparatus and the Mitochondria," *Quart. Journ. Micr. Sci.*, 1919, vol. lxiii, pp. 445-493.
[9] *Idem.* "The Cytoplasmic Inclusions of the Germ Cells. Part VII. The Modern Technique of Cytology," *Quart. Journ. Micr. Sci.*, 1920, vol. lxiv, p. 3.
[10] Golgi, C. "Intorno alla struttura delle cellule nervose," *Boll. Soc. Med. Chir. di Pavia*, 1898, pp. 53-63; or *idem*, "Sur la structure des cellules nerveuses," *Arch. ital. de Biol.*, 1898, t. xxx, p. 60.

[11] Golgi, C. "Sur une fine particularité de structure de l'épithélium de la muqueuse gastrique et intestinale de quelques vertébrés," *Arch. ital. de Biol.*, 1909, t. li, p. 213; *Arch. per le Sci. Med.*, 1909, t. xxxiii, p. 1.
(For full bibliography of Golgi's work on this subject, see this article, p. 215.)

[12] Holmgren, E. "Studien in der feineren Anatomie der Nervenzellen," *Anat. Hefte*, 1900, Bd. xv, S. 1-90.

[13] *Idem.* "Einige Worte über das 'Trophospongium' verschiedener Zellarten," *Anat. Anz.*, 1902, Bd. xx, S. 433-440.

[14] *Idem.* "Trophospongium und Apparato reticolare der spinalen Ganglienzellen," *Anat. Anz.*, 1914, Bd. xlvi, S. 127-138.

[15] Kopsch, F. "Die Darstellung des Binnenetzes in spinalen Ganglienzellen und anderen Körperzellen mittels Osmiumsäure," *Sitzungsber. d.k. preuss. Akad. d. Wiss.*, Berlin, 1902, S. 929.

[16] Legendre, R. "Recherches sur le réseau interne de Golgi des cellules nerveuses des ganglions spinaux," *Anat. Anz.*, 1910. Bd. xxxvi, S. 207-215.

[17] Marcora, F. "Di una fina alterazione delle cellule nervose del nucleo di origine de grande ipoglosso consecutiva allo strappamento ed al taglio del nervo," *Boll. Soc. Med. di Pavia*, 1908, t. xxii, pp. 134-137.

[18] *Idem.* "Ueber die Beziehungen zwischen dem Binnenetze und den Nisslschen Körperchen in den Nervenzellen," *Anat. Anz.*, 1910, Bd. xxxv, S. 65-69.

[19] *Idem.* "Ueber die Histogenese des Zentralnervensystems mit besonderer Rücksicht auf der innere Struktur der Nervenelemente," *Folia Neuro-biol.*, 1911, Bd. v, S. 928-960.

[20] Nissl. "Ueber die Veränderungen der Ganglienzelen am Facialiskern des Kaninchens nach Ausreissung der Nerven.," *Allgem. Zeitschr. f. Psychiatr.*, 1892, Bd. xlviii S. 197-198. (Quoted by Van Gehuchten, 30.)

[21] Nusbaum, J. "Ueber den sogenannten inneren Golgischen Netzapparat und sein Verhältnis zu den Mitochondrien, Chromidien und anderen Zellstrukturen im Tierreich (Zusammenfassendes Referat)," *Arch. f. Zellforsch.*, 1913, Bd. x, S. 359-367.

[22] Penfield, W. G., 1920. "On the Relationship of the Golgi Apparatus and Holmgren Trophospongium in Nerve Cells: Comparison during Retispersion." (To appear shortly.)

[23] Ramon y Cajal, S. "Estudios sobre la degeneración y regeneración del systema nervioso," 1914, t. ii, Madrid.

[24] *Idem.* "Algunas variaciones fisiológicas y pathológicas del aparato reticular de Golgi," *Trab. Lab. Invest. Biol. Univers.* Madrid, 1915, t. xii, p. 2.

[25] *Idem.* "Variaciones fisiológicas del reticulo de Golgi en algunos elementos epiteliales y mesodérmicos," *Bol. de la Soc. Espanola de Biol.*, 1915, March.

[26] Sjövall, E. "Ein Versuch, das Binnennetz von Golgi-Kopsch bei der Spermato und Ovogenese zu homologieren," *Anat. Anz.*, 1906, Bd. xxviii, S. 561-579.

[27] Sherrington, C. S. "Mammalian Physiology," Oxford, 1919.

[28] Sherrington, C. S., and Laslett. E. E. "Remarks on the Dorsal Spino-cerebellar Tract," *Journ. of Physiol.*, 1903, vol. xxix, pp. 189-194.

[29] Van Gehuchten, A. "A propos du phénomène de chromatolyse," *Bull. Acad. roy. de Méd. de Belg.*, 1898, 4me sér., t. xii. pp. 222-230.

[30] *Idem.* "Anatomie du système nerveux de l'homme," 4th edition, Louvain, 1906.

Section of Neurology of the Royal Society of Medicine.

President—Dr. W. Aldren Turner, C.B.

Dr. S. A. Kinnier Wilson read a paper "On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits,"[1] which appears on p. 220 of this number. A discussion followed, in which took part :—

DISCUSSION.

Dr. H. C. Bazett: I have found Dr. Kinnier Wilson's paper of great interest since his cases show such striking similarities with the decerebrate animals described by Professor Sherrington, and yet in some respects no less definite contrasts. I have recently been employed in Professor Sherrington's laboratory in developing a technique by which cats may be kept under observation for two or three weeks, even after removal of all nervous tissue lying anterior to the posterior colliculi. These chronic decerebrate animals differ only slightly from the acute preparations, but in some ways they provide a better standard for comparison with clinical material, and their importance lies in the fact that the localization of the lesion can be accurately determined.

The main similarities are that in these animals too the extensor rigidity may be maintained for days or weeks, that this position may alternate with positions of flexion, and that tonic fits with marked opisthotonos may be occasionally seen soon after the operation. A common result is to obtain an animal, which keeps up usually a position of extensor rigidity, but which after a few days also shows occasionally positions of flexion, and this flexion is sometimes flaccid but may be rigidly maintained. It is also common for a preparation, which, when approached quietly (since these animals are often very sensitive to auditory stimuli), is seen to be lying in a flexed position, to assume a position of extensor rigidity as soon as any examination is attempted. In some cases this may be induced by mere auditory stimuli.

The occurrence of tonic fits is comparatively rare, and is limited to the first few hours after the operation, except in some cats in which meningitis has developed. In the early cases the opisthotonos is extreme, the respiration is generally slow but may be fast and shallow, and occasionally with a slow respiratory rate the fits themselves have a respiratory rhythm. These animals are very different from the better preparations, in which the respiratory rate is normal, and they rarely survive long if the fits are at all severe. The operation often causes a certain amount of hæmorrhage below the tentorium into the subarachnoid space, but this hæmorrhage seems to have been

[1] At a meeting of the Section, held May 13, 1920.

particularly noticeable in the animals which have shown tonic fits. In any case the origin of these fits in the nervous system below the corpora quadrigemina is confirmed.

The main contrasts are that these preparations never show any athetoid movements or tremor, that they never show a pyrexia but a complete loss of temperature control with a rapid fall of body temperature, unless they are artificially kept warm.

They have a temporary glycosuria which disappears within about three days after the operation; they show brisk corneal reflexes, while vocalization in the form of purring, growling, and very rarely mewing may occur.

A few of the animals have survived sufficiently long to allow degeneration to take place, and the remaining parts of the central nervous system are being examined for me by Dr. W. G. Penfield, so that further data should shortly be available. It is at any rate clear that decerebrate rigidity persists even after the rubrospinal tract is mostly degenerated, so that if the red nucleus is responsible for the rigidity it must be the lower part of this nucleus that is concerned: and it is established that the rigidity is not due to the excitation of nerve fibres divided in the operation, since in this case the rigidity could not be maintained after the degeneration of the fibres.

Dr. WALSHE: While we may agree with Dr. Wilson that tonic or postural "fits" are probably of the nature of accentuations of Jackson's cerebellar attitude, I do not think that Jackson's view of the origin and relationship of these phenomena has always been correctly interpreted by those who have at various times referred to his work. In this respect, I notice that Dr. Wilson has adopted the usual view that Jackson regarded both the persisting attitude and the "fits" as identical in origin. This is not what Jackson held, and I have always been puzzled to understand how it was that in this connection he departed from his usual teaching in respect of rigidity and ascribed it in this instance to *cerebral* influx. If we refer to the papers in which he described his cases of cerebellar tumour with "tetanus-like seizures" and cerebellar attitude, which were reprinted in vol. xxix of BRAIN in 1906, we find (p. 432) the statement that "the patient's persisting attitude (cerebellar paralysis with rigidity, that is, a cerebellar attitude). . . . was, in general appearance, like that in the tetanus-like seizures, but the process of causation of the two conditions is, in my opinion, very different." And again (on p. 441), "the rigidity fixing the attitude is the result of cerebral influx, that influence being no longer antagonized, or being less antagonized, by cerebellar influence. Tetanus-like seizures are, I submit, owing to changes of instability set up in nerve cells near the tumour, owing to a cerebellar discharge lesion; the tetanus-like seizures are owing to occasional intense discharges of this persisting discharge lesion." For various reasons it is extremely difficult to believe that phenomena so closely alike in their characters can differ widely in origin, or that the persisting attitude is an expression of cerebral influx. It is far more probable that, whether we accept their identification with decerebrate rigidity or not, both have a common cause, as Dr. Wilson believes. However, it is not correct to attribute this view to Jackson.

Publications Recently Received.

Cerebrospinal Fever. By Michael Foster, M.A., M.D., and J. F. Gaskell, M.A., M.D. Pp. 222. Cambridge University Press, 1916.

In this volume the authors give a systematic account of cerebrospinal fever, based upon their extensive experience in the epidemic of 1914-15. They show reasons for preferring this term to the longer one in common use, epidemic cerebrospinal meningitis. The book deals fully with every aspect of the disease, but is admirably concise. The clinical section is particularly well arranged, and we have seldom read a clinical description at once so complete and so lucid. The chapters devoted to the evolution and course of the malady and its diagnosis are excellent and prepare the reader for the later chapters dealing with the various clinical forms of cerebrospinal fever, so that these do not have the arbitrary and artificial precision so common in clinical classifications.

The authors have attempted, as far as possible, to correlate the clinical manifestations with the underlying pathological lesions. They regard the essence of the disease as a true infection of the cerebrospinal system, causing death in fatal cases by the intensity of the pathological process, and not as a meningococcal septicæmia in which the blood-borne infection is the potent factor.

Both toxæmic and physical factors, however, play a part in the disease process. Thus, they suggest that the purulent exudate overlying the brain acts both by poisoning and by compressing the brain. It is probable, however, that some of their suggestions in this respect have but little foundation. For example, they regard the characteristic head retraction as a manifestation of increased intracranial tension. If this were so, we should expect to find head retraction a common symptom in cerebral tumour, whereas it is of extreme rarity. It is more likely that it is an expression of the extensor rigidity which Jackson described under the name of cerebellar attitude, or it might even be a reflex spasm of the neck extensors from meningeal irritation. The work of Cushing and his school upon the pathways of the cerebrospinal fluid and upon the production and nature of internal hydrocephalus was not known in this country at the time this monograph was written, and it throws light upon several points raised by Foster and Gaskell, and perforce left unanswered by them.

In the chapter devoted to prognosis they note the rarity of residual paralytic phenomena and the absence of mental defect as a sequel of cerebrospinal fever.

They hold that the essence of all treatment lies in the adequate drainage of the subarachnoid space by repeated lumbar punctures, long persisted in in chronic cases. The object of each puncture should be complete evacuation of the excess of fluid, which is always found, except in a few chronic suppurative cases.

The series of coloured plates, at the end of the text, scarcely do justice to this valuable monograph, which must become the standard work of reference on this disease in the language. There is a complete bibliography.

Instinct and the Unconscious. A Contribution to a Biological Theory of the Psycho-neuroses. By W. H. R. RIVERS, M.D., D.Sc., LL.D., F.R.S. Pp. 252. Cambridge: University Press, 1920.

After many years spent in teaching psychology and the physiology of the senses at Cambridge, Dr. Rivers turned his attention to anthropology, and is well known as the author of the "History of Melanesian Society," and for his "Studies on Kinship and Social Organization." After the outbreak of war, he was engaged in treating wounds of peripheral nerves, but soon became attached to Maghull Military Hospital, and later to Craiglockart War Hospital, where he had unrivalled opportunities for gaining experience of the psychoneuroses of war. Later, he was appointed to the staff of the Royal Air Force Hospital at Hampstead: here, and on the aerodrome at Hendon, he accumulated an unique experience of the temperament and neuroses of the flying man.

The results were embodied in a series of papers published during the last three years: these form six appendices at the end of this book, and contain an account of "A Case of Claustrophobia," "The Repression of War Experience," "War Neuroses and Military Training," and an excellent short note on "Wind-up."

The bulk of the work, however, is occupied in an attempt to bring the abnormal phenomena of mental life into harmony with the processes and activities with which we are now familiar on the physiological level. Such subjects as the unconscious, suppression and inhibition, the nature of instinct, the danger instincts and suppression, dissociation, suggestion, sleep, the substitution neuroses, regression and sublimation, are dealt with in a series of short chapters. These are clear and admirably written, and will be of the greatest value to all who are interested in the nature and workings of the unconscious. Moreover, the theory that instincts are suppressed primitive mental activities, which may be either held entirely in check or used in part, is a brilliant addition to our knowledge. Dr. Rivers has much to say on the mechanism of suppression and dissociation, which is equally luminous. It is impossible in a short space to give an adequate account of the originality and interest of this book, which deserves repeated reading.

Injuries of the Peripheral Nerves. By HENRY S. SOUTTAR, C.B.E., F.R.C.S., and EDWARD W. TWINING, M.R.C.S., L.R.C.S. Pp. 152 with 30 figures. Bristol: John Wright, 1920.

This is an admirable book clearly written and excellently illustrated. No attempt is made to enter into theoretical questions, but the gross effects produced by division of each of the larger nerve trunks are considered in detail, with especial relation to the site and nature of the injury. As might be

expected from a surgeon of Mr. Souttar's wide experience, the various operative procedures are clearly described with the help of admirable drawings. It is encouraging to find so close an agreement between the conclusions arrived at independently by the authors of this work and the Committee of the Medical Research Council. It shows that, at any rate in England, a harmonious body of opinion is gradually growing up on the diagnosis and treatment of peripheral nerve injuries. We strongly recommend this practical book to all who are interested in the problems of the surgery of nerve lesions.

The Diagnosis and Treatment of Peripheral Nerve Injuries. [Report of the Committee upon Injuries of the Nervous System.] Published by the Medical Research Council. Pp. 59. London: His Majesty's Stationery Office, 1920.

An attempt has been made in this pamphlet to lay down the general principles which should guide diagnosis and treatment in cases of injury to the peripheral nerves. But, in addition, the main nerve trunks are considered individually, and the various difficulties, due to peculiarities in their constitution and function, are considered in each instance. All theoretical questions are avoided and the advice offered, or procedure described, is given in the simplest terms.

Part I deals with nerve injuries in general, treating succinctly of diagnosis, non-operative treatment, the nature of the various operations to be performed, the signs of returning function and the phenomena of pain. In Part II, the signs and symptoms of injury, together with the methods of treatment to be adopted, are considered for each of the main nerve trunks in turn. In Part III the histological appearances are described as they appear under different conditions of injury. Finally, the treatment of painful states, such as "causalgia," and the factors which produce them are dealt with briefly. The Report is intended as a succinct guide for those who are engaged in treating nerve injuries resulting from the war, or in assessing the various degrees of disability they may produce.

Diagnosis and Treatment of Brain Injuries with and without a Fracture of the Skull. By William Sharpe, M.D., Professor of Neurologic Surgery, New York Polyclinic Medical School. Pp. 757. Philadelphia and London: Lippincott, 1920.

The author deals with methods of diagnosis and describes in detail the signs and symptoms, treatment and progress of individual patients with various forms of brain injury. He rightly insists on the importance of considering the form and situation of the lesion of the brain, as opposed to the injury of the skull. For, in treating patients with head injuries, the important factor is not

the presence or absence of a fracture, but rather the nature of the intracranial damage and increased cerebral tension. Stress is also laid on the necessity for considering the future condition of the patient, as well as preservation of his life.

The book is divided into three main parts :—

(1) General conditions, such as pathology, diagnosis and treatment, including operative technique ; (2) Brain injuries in adults, both acute and chronic ; (3) Brain injuries in new-born babies and children. Full records are given of 195 cases and the illustrations are good.

It is urged that the enormous mortality, often 50 per cent., following operative treatment in the acute stage, is largely due to the inopportune time chosen by surgeons for interference. The two periods during which operation is distinctly contra-indicated are, firstly, during the early stage after the injury, when the patient is suffering from shock, and, secondly, after signs have appeared of medullary compression. In the author's opinion, expectant palliative treatment is successful in two-thirds of the cases, excluding depressed fracture of the vault ; but in the remaining third, where there is considerable increase of intracranial pressure, the skull and dura should be opened, preferably in the subtemporal region. We note with surprise that all methods for closing cranial defects are condemned, and no mention is made of the distressing symptoms which are liable to develop in patients with large openings in the skull.

The Medical Problems of Flying, including Reports I to VII of the Air Medical Investigation Committee. Published by the Medical Research Council. Pp. 272. London : His Majesty's Stationery Office, 1920.

During the course of the war, flying was gradually carried on at greater and greater heights, so that by the autumn of 1916 " mountain sickness " in various degrees gravely increased the difficulty and dangers met by flying officers in the course of their work in the air. Much experimental work was undertaken by individual medical officers, expert physiologists, and civilian physicians, and a series of special reports were published under the ægis of the Medical Research Committee. The papers which comprise this volume contain much that is both directly and indirectly of interest to neurologists. They deal with the reaction of pilots and observers to diminish oxygen pressure, as measured by Professor Dreyer's method, and by the various tests introduced by Colonel Flack. The papers on " Methods used in selecting Candidates for Flying," " On the Sense of Stability and Balance in the Air," on " Mental Aptitude for Aviation," and on " Reaction Time Tests carried out on Flight Cadets," all bear on the functions of the central nervous system in an unexpected and novel manner. Some of the tests described are worthy of more extended application to neurological practice.

Les Maladies nerveuses. Cours professé à l'Université de Louvain. By the late Professor A. VAN GEHUCHTEN. Pp. 845. Louvain: Librairie Universitaire, 1920.

Professor van Gehuchten was preparing to publish the course of lectures delivered by him in the University of Louvain, when the work was stopped by the outbreak of war. The first four hundred pages were already in print, the remainder was in manuscript. The plates prepared to illustrate this book were burned by the Germans, but they have been excellently reproduced from photographs, and a large number of diagrams show the anatomical relations necessary for understanding the signs of the various diseases described. The whole has been passed through the press by the filial piety of Dr. Paul van Gehuchten in memory of his distinguished father.

As the title indicates, this book is intended for students, not for neurologists, and probably for this reason all references are omitted. Organic diseases are classed under such headings as peripheral nerves, muscles, spinal cord, mid-brain, &c. This necessitates somewhat summary treatment, for all morbid conditions are described in terms of anatomy, and little attention is paid to disturbance of function. The last section of the book is devoted to "functional nervous disorders" grouped under the headings of chorea, paralysis agitans, epilepsy, hysteria, neurasthenia and traumatic neuroses. It is a profound misfortune that the lamented author died before his work was completed, and without having the opportunity to embody the great advances in neurology made during the last six years.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I to XXIII inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

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BRAIN.

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LEFT-HANDEDNESS AND MIRROR WRITING, ESPECIALLY AMONG DEFECTIVE CHILDREN.

BY HUGH GORDON, M.A.

Chapter I.—Introduction.

The following observations and experiments were made, unless otherwise stated, in special schools (for mentally defective children) in London and Middlesex.

The Elementary Education (Defective and Epileptic Children) Act, 1899, defines the children, for whom these schools are provided, as "not being imbecile and not being merely dull or backward, are defective, that is to say, children, by reason of mental or physical defect, are incapable of receiving proper benefit from the instruction in the ordinary public elementary schools, but are not incapable, by reason

of such defect, of receiving benefit from instruction in special classes or schools, &c."

The investigation originated from an observation made in April, 1918, during the inspection of one of these special schools. In one of the classes nine (37 per cent.) of the children out of twenty-four were writing with their left hands, and doing most of their work left-handedly. At other mental defective schools the percentage of left-handed children appeared to be very high; and head teachers frequently reported that there was a large number of left-handed children in their schools. As the percentage of left-handed children was undoubtedly much higher in mental defective than in the ordinary elementary schools, an attempt was made to determine as accurately as possible their number, and to include in this number those who were originally left-handed, but who had been converted into right-handedness in many of their activities by the pressure brought to bear on them by parents or teachers.

A left-handed child was taken to be one who would use the left hand in preference to the right in such activities as are usually carried out by the right hand in the case of right-handed children. It was originally considered important that the tests should be in such things as might have passed unnoticed by parents and teachers, and for this reason might have remained unchanged. Owing to little time being available at first for the investigation, it was necessary that the tests should be of the simplest character and that they could be applied rapidly, and to whole classes. A great variety of experiments have been made, and only those tests which gave consistent results have been retained. The methods used and the results obtained will be given later.

The high percentage of left-handed children in mental defective schools does not appear to have received any special attention. The following references, however, are of interest in connection with the subject.

Ballard in an investigation on "Sinistrality and Speech" in 1912, notices a higher percentage of left-handed children in mental defective schools than in ordinary elementary schools, i.e., 6·5 per cent. against 4·1 per cent.[1] In both percentages he includes sinistrals (left-handed writers, &c.), and what he calls dextro-sinistrals (left-handed children who write with the right hand).

Smedley [28], 1899, in some experiments on the "strength of grip"

[1] These numbers were obtained from reports, &c., from the schools. In his own investigations he gives the percentage in ordinary schools as 2·8 per cent.

of right and left hand concludes that "dull pupils are more ambidextrous than average, and average than bright pupils; while the John Worthy school boys (incorrigibles, truants, &c., Chicago), are more ambidextrous than the dull pupils of the regular schools."

In other experiments on children, e.g., in the "tapping tests" (i.e., tapping as rapidly as possible with the right and with the left hand in a given time), Smedley again comes to the same conclusion: "left hand efficiency nearly approaches right hand efficiency in the case of dull and backward children." This conclusion, however, is not confirmed by Bolton. It must not, however, be forgotten that, in the case of left-handed children and adults who write with the right hand, the difference between the efficiency of the hands is likely to be less than when a person is simply right-handed or left-handed. In fact, during the present investigation there appeared to be little evidence of real ambidexterity; there were a few exceptional cases in which either hand was used indiscriminately. The children were of very low-grade type, and their lack of so-called "dexterity" was very marked in the case of both hands.

In "Ambidexterity," J. Jackson [13] refers to the opinion that the insane have a great percentage of "partial ambidexters" in their ranks, and that there is a high percentage of left-handed among criminals; also that left-handed children have been looked upon as somewhat daft, hence nurses and parents did their best to make them right-handed.

Lombroso, however, states that the percentage of left-handed among lunatics does not greatly differ from that among normal people, but that it is much higher among criminals (*North American Review*, 1903). Mayet (1902) found a high percentage (16 to 30) of ambidextrous and left-handed among idiots and epileptics. Lombroso himself considered left-handedness as a sign of degeneracy.

Ballard in 1912 [5] and W. Franklin Jones [14] in 1918 both came to the conclusion that stammering in a high percentage of cases is closely associated with the "transfer" of congenital left-handers to right-handedness in writing and vice versa.

General attitude of teachers towards left-handed children.—In mental defective schools, teachers as a rule after some attempts at converting left-handed children into right-hand writers, and if these attempts are not very successful, leave them to their own devices. Knitting and sewing however are treated differently in the majority of cases, owing to the difficulty experienced by teachers in teaching these subjects left-handedly. As a matter of fact very few were found

to knit or sew left-handedly. Probably the lack of adaptability of mentally defective children renders it extremely difficult to bring about these changes (especially in writing); and this may explain why teachers in these schools have found it advisable to leave the children alone. In one school the teachers try to make all the scholars right-hand writers. In this school twenty-eight (46 per cent.) out of a total of sixty-one were found to be left-handed by the various tests described later. Of these twenty-eight, sixteen were well known to have written or to have drawn with their left hand at one time or other. In connection with this point it must be remembered that few children are sent to these special schools before the age of eight, and it is therefore highly probable that most of them have experienced some pressure to make them write with their right hand. Clearly also the pressure had been insufficient to bring about the change. Of these twenty-eight, thirteen are reported to have some "speech difficulties," and two to be "defective in speech," one to "stutter," and two to "stammer" at times. Too much stress must not be laid on such defectiveness, as difficulties in speech apart from left-handedness are not uncommon among this class of children. At another school the children are especially trained in ambidexterity and even in writing with both hands.

In ordinary elementary schools, teachers generally assert that at the present time little or no pressure is brought to bear on left-handed children. As a matter of fact most left-handed children are subjected to some pressure either by teachers or parents, but generally unaccompanied by any harshness. A great many of these left-handed children are of a highly nervous temperament, and in many cases this nervousness does not appear to be due to a forced change of hands, or to an unsuccessful attempt to convert them into right-hand writers. It is however difficult to say anything very positively, as it is almost impossible to ascertain the true facts of each case.

Chapter II.—Tests and Methods of Examination.

§ 1.—*Tests.*

A great many experiments have been made, e.g., kicking, tying knots, the use of the hammer and saw, &c., but in most of these many difficulties were encountered, and in some it was found that the results were inconsistent with the known facts. In kicking a ball for example, it was often found that thoroughly left-handed boys kicked with the

right foot, and in the case of girls and younger children no reliance could be placed on the results, as they frequently kicked with the foot which happened to be in the most convenient position when they reached the ball.

Originally all children, who could write their names, were asked to make the attempt with their left hand, as it was expected that the result might indicate those who had some skill with this hand. The instructions were very simple ; the children were told to take their pencils in their left hand and to hold them up. In this way it could be seen that everyone had them in the left hand. They were then asked to write their names with that hand, on a slip of paper that had been given them. After a little hesitation nearly everyone made the attempt. It was interesting to note the difficulties they experienced, and how they overcame these difficulties. Many began by holding the pencil four or five inches from the pointed end and attempted to write with an arm, instead of a wrist or finger movement. As a rule however they gradually changed to a more suitable position and movement. It was remarkable—considering the class of children—how well they wrote their names as a rule. One peculiarity was immediatey noticed —namely "mirror writing." As this type of writing was found to be prevalent among mental defectives, a special investigation was made into the matter, the results of which are given separately.

After prolonged experiments the following tests were found to be the most reliable and most consistent with known facts. In fact very seldom was a known left-handed child missed, and very frequently left-handed children, unknown to the teacher, were discovered who on careful inquiry were found to have been left-handed in their earlier school career.

(*a*) *Rubbing and throwing test.*—The names of the children in a class were written on a sheet of foolscap and the experimenter went round to each child with a duster and placed it on the desk, asking the child to give the desk a good rub. As a rule the child seized the duster, and rubbed the desk vigorously with the right hand ; the left-handed child, however, seized the duster and rubbed the desk with the left hand. It was very necessary to look at the non-rubbing hand, as not infrequently a right-handed child with a penny or handkerchief in the right hand rubbed with the left hand. Those who rubbed with the left hand were immediately asked to stand at the back of the room, and to catch the folded-up duster and throw it back. In infant schools these two tests were remarkably successful. The test was very quickly

carried out; and throwing with the left was found very often to be associated with left-hand rubbing. The teacher, who had the list of names, entered "L" in the columns headed "Rub" and "Throw" in the case of those who did it with their left hand. When a child rubbed with the left hand and threw with the right, he was again asked to rub the desk, care being taken to place the duster so that it could be taken more easily with the right. If he still persisted in rubbing with the left the letter "L" was entered, but, if he now changed to the right, the letter was entered but a line was drawn through it. Interesting cases were continually met with when a child was asked to catch the duster; some immediately placed the left hand behind their backs or held their left arm stiffly down with their fist clenched and put the other hand well in front. Some of these actually rolled up the duster with the right hand, still holding the left arm down or behind them. It was evident they were not acting in a natural way, that they were in fact inhibiting the natural action.

Among the older children in ordinary schools, i.e., those above Standard II (about $8\frac{1}{2}$ years of age) and in the elder boys and girls, special schools (over 12), the rubbing and throwing tests were not so successful for various reasons. Often those who were being tested soon understood the object of the test; some wished to conceal their left-handedness, others thought that the trial was to find out those who could throw well with the left hand. It is important in these and the following test that both should be made before any knowledge of their exact purpose has been gained.

As an example of the result of these two tests the following is given :—

Infant school, the lowest class, ages $4\frac{1}{2}$ to $5\frac{1}{2}$, forty-one boys and girls. Seven were found to rub and throw with the left hand and one to rub with the left but throw with the right. Three days afterwards the whole class were asked to draw anything they liked, and the seven drew with the left hand, and all the others with the right.

(*b*) *Throwing tests.*—A test for older boys and girls. A waste-paper basket was placed about four or five yards from a line marked on the floor. After explaining that it was wished to discover those who could throw the straightest, by seeing who could throw a knotted duster into the basket from the line marked on the floor, the experimenter sat at the table with the list and called out in order the names of the boys or girls, and noted those who threw with the left hand. In this way the test was carried out without the "subjects" having any knowledge of the real object of the experiment.

The results of this test in three elder boys' schools (for mentally defective) are given below :—

School A.—Classes 1 and 2 : thirty-one boys, ages 13 years to 16 years 8 months. Five boys (and five boys only) threw with the left hand ; all of these and no others were conclusively proved to be left-handed. Of these, two always write with the left hand, and one with the right ; the remaining two write with the right, but used to write at one time with the left.

School B.—Classes 1 and 2 : forty-six boys. Four boys threw with the left ; of these three were proved to be left-handed, and they write with the left hand. One had some clear indications of left-handedness, but writes with the right hand. Of the rest, three who threw with the right, each had some slight indications of left-handedness, but insufficient to be noted as left-handed : they all write with the right hand.

School C.—Classes 1 and 2 : sixty-one boys, ages 11 years 9 months to 15 years 10 months. Six boys threw with the left ; of these five were proved to be left-handed ; four of them write with the left and one now writes with the right, but used to write with the left at one time. Of the remainder, two boys who threw with the right, one was clearly left-handed ; he draws and writes with the left hand : one had some indications of left-handedness, and is much stronger with the left-hand grip ; dynamometer, left 59, right 45.

From the above facts it is evident that left-hand throwing is as a rule a sign of general left-handedness. It is not unlikely however that there may be exceptional cases, where this diagnosis may not be correct, e.g., injury to the right hand or arm at a critical age. On the other hand it is equally clear that throwing with the right hand is not always associated with right-handedness. Parents do undoubtedly effect the change, often at an early age.

(*c*) *Scissors test.*—The scissors in general use are made for right-handed people. In attempting to cut with the left hand anyone naturally holds them in the same way as he has learnt to do with the right hand, and if the two blades are loosely attached these are in consequence forced apart instead of together. The cutting of paper, linen, &c., is impossible under these conditions, especially if the scissors are not very sharp. This test seems to be a most reliable one with children, and especially with dull and backward ones. With adults however, it is different, as a great many have learnt to cut their nails with the scissors in their left hand. Many do not clearly understand how they do it, and a fairly high percentage cannot do it at all. It is often very difficult to teach them, and frequently after having learnt they find they have lost the knack, by merely cutting a piece of paper again with the right hand. Some left-handed children however can cut

a piece of paper only with their right hand with these scissors, but as a rule they can cut with the left or with both.

Children who can cut with these scissors with the left hand may be divided into two classes:—

(1) Those who can cut equally well with left and right hands.

(2) Those who can cut with the left but not with the right.

Among the youngest children—about 5 years of age in the ordinary elementary school, and about 7 to 8 in the mental defective school, there are many who cannot cut with these scissors with either hand, but are quite capable of doing so with the ordinary scissors in use in the schools.

A great many tests have been made with these specially prepared scissors, and the following results indicate their reliability. The scissors are merely loosened, and the cutting edge rendered less sharp by filing.

In the case of 318 children in mental defective schools, who cut with the left hand—

(i) 203 cut with left and right; of these, 172 were certainly left-handed (about 86 per cent.), eight had other indications of left-handedness, twenty appeared to be right-handed, and one appeared to be ambidextrous: the remaining two had been taught at home.

(ii) Fourteen cut with the left and could cut a little with the right; of these, fourteen were left-handed (100 per cent.).

(iii) 101 cut with the left only; of these, ninety-seven were left-handed (about 97 per cent.), two were probably left-handed, and two appeared to be right-handed, and no explanation of their skill was found.

Put in another way, out of 318 children who cut with the left hand 283 (89 per cent.) were left-handed, and nine (2·8 per cent.) were probably left-handed.

Below is given the percentage of left-handed children who cut with the left only, or with left and right hands:— .

Number of left-handed children	Cut with left and right	Cut with left only
244	102 (41·8 per cent.) ..	78 (32 per cent.)

i.e., about 74 per cent. of the left-handed could cut with the left hand, and roughly speaking about a quarter could not cut with the left hand. Only very young right-handed children are unable to cut with these scissors with the right hand. All left-handed children, therefore, are not able with these scissors to cut with their left hand; in fact, only 75 per cent. can do so.

From these facts it is evident that (i) if a child can cut with these scissors with the left hand, and only with the left hand, he is nearly

certain to be left-handed, i.e., to use the left hand as the dominant hand in many of his activities.

(ii) If a child can cut equally well with both hands, he is probably left-handed.

(iii) If a child cannot cut with his left but can cut with his right hand, it must not be inferred without further evidence that he is not left-handed in many of his activities.

Attempts were made to discover how some of the markedly right-handed children had learnt to cut equally well with their left hand. In many cases no explanation could be found, and this is what might have been expected, as it is often difficult to obtain information from children found in mental defective schools. The following cases are interesting and are not unimportant when the whole question of left-handedness comes to be considered later on in this paper.

One boy explained that he never could cut with his right, and that his brother, who always cuts with his left and is left-handed, taught him. This boy is still unable to cut with his right hand, as he holds the scissors exactly in the same way as he does with the left, and so forces the two blades apart. In two cases the children who could cut equally well with either hand had been taught at home.

In elementary schools: (i) A girl, aged about 11 years, cut with both left and right hands, but asserted she had never been left-handed. On inquiry the head teacher of the infant school from which she came remembered quite distinctly that at first she used to write with her left hand; (ii) a girl, aged about 7 years, cut equally well with either hand, but in every other test gave no indication of left-handedness. It was then remembered that the girl had broken her right arm some time ago, and had come to school for three months with the arm in a sling. She was found to be able to write her name equally well with either hand.

(*d*) *Picking-up test.*—A large number of left-handed children pick up anything on the ground with their left hand, provided their attention is concentrated on the picking up and not on the question of the hand they are going to use.

Those to be tested were arranged in a line and a small piece of paper was placed exactly in front of each. They were told that there was to be a race to see who could pick up the paper first and hold it up.

It was found best to say: "Stand up straight with your arms down," and immediately afterwards, "Go." In many cases it was noticed that some left-handed children, when getting ready, placed their left hand

behind them, apparently to force themselves to pick up with the right. If however they were made to stand up straight with arms held down and started immediately, the left-handed picked it up quite naturally, and with the left hand. Sometimes children started to pick up with the left, and then suddenly changed to the right. Whole classes have been tested ; about ten or so were tested at a time in the hall, and those who were holding the paper in the left hand kept back ; these, when all the class had been tested, were each given a piece of paper and asked to place it on the floor, and they were then raced again. Under these conditions it was found that right-handed children did not often pick it up with the left, but that in a certain number of cases the left-handed picked it up with the right. The test by itself may not be of much value, but taken with the other tests it is by no means unconvincing.

(*e*) *Winding test.*—As a rule in winding cotton on a reel, one hand is used to hold the reel and the other to wind. Among right-handed people the winding is done by the right hand, and in the case of the left-handed by the left. Sometimes the winding is carried on by the hand holding the reel, in either case however the winding is done by the dominant hand. A few children and a good many adults move both hands while winding the cotton. In such cases a simple apparatus can be used to prevent the movement of the holding hand, and the time can then be taken for winding a yard of string by each hand by means of a stop-watch.

The following experiments show a much greater speed with one hand than the other : a left-handed boy, right = 5 seconds, left = 3 seconds ; a right-handed boy, right = 5 seconds, left = 7 seconds.

One fact is very noticeable—the winding by the dominant hand is done chiefly by a wrist movement, whereas when done by the non-dominant hand, the action is from the elbow or even shoulder, i.e., an arm movement. Notice has already been called to a similar movement in writing with the non-dominant hand, and further reference will be made to similar peculiarities in the "line test." It appears therefore that the tendency of the uneducated and unpractised hand is that of an arm and not of a wrist and finger movement.

(*f*) *Line test.*—This is a quantitative test, but it was intended merely to give an indication of left-handedness, especially in cases of doubt. It can be carried out very rapidly and a whole class of even sixty can be tested at the same time.

Slips of paper were distributed to the class, on which to practise

making as quickly as possible a number of separate vertical lines of about $\frac{1}{4}$ to $\frac{1}{2}$ inch in length, first with the right and then with the left hand. The blackboard was used to demonstrate exactly what was to be done. After one or two minutes' practice, new slips of paper were given out, and it was explained that there was to be a race to discover who could draw the greatest number of lines in a given time. The children were told to write their names on the slips, and then to hold up their pencils in their right hand. It was explained that at the word "go" they were to start making lines as quickly as possible and to continue until stopped after fifteen seconds. At the end of fifteen seconds the following instructions were given rapidly: "Stop; put down your pencils, and turn over the paper." Exactly the same instructions were given in the test with the left hand. The test always aroused interest and amusement, especially when the left hand had to be used. The same procedure was used in mental defective and in ordinary elementary schools.

The results were often very convincing, but it was always necessary when the test indicated left-handedness to scrutinize carefully the lines made by each hand, as sometimes left-hand speed was clearly due to less care being taken in drawing the lines.

In some cases, however, especially among children in mental defective schools, no reliance could be placed on the results: many of these children were in fact quite unable to concentrate their attention even for five seconds; and after making a few lines these stopped and looked round to see how the others were getting on. It was interesting to see how nearly every one was anxious to do his best and show his skill. As has already been mentioned, the arm movement of the non-dominant hand was very noticeable. The test was found to be most useful among ordinary children of about 11 years of age and upwards, and among adults.

There was a great variability in the number of lines drawn, but it was evident that as a rule the older and more intelligent the child the greater was the number of lines, as might naturally have been expected. The proportion between the number of lines made by the right and by the left hand varied in every possible way, but in right-handed adults the number made by the right was always at least 29 per cent. more than that made by the left hand. Left-handed adults, who have from childhood written with the right hand, as a rule did about 6 per cent. more with the right than the left.

The results of some of these tests are given in percentages of the

number of lines made by the right hand over that made by the left. If a greater number is made by the left hand the percentage is marked by a minus sign, e.g., 50 per cent. indicates that the number of lines drawn in fifteen seconds by the right hand was 50 per cent. more than the number drawn by the left hand, and 50 per cent. (–) indicates that the number drawn by the left hand was 50 per cent. more than that drawn by the right. A higher percentage would therefore point to greater difference in "handedness."

(i) Line test—percentages for right-handed adults : 29 (two cases) ; 30 (two) ; 32 ; 36 (four) ; 39 ; 41 ; 43 ; 48 ; 51 ; 59 ; 68 ; 85 ; 96.

Note.—There was also a case of 21 per cent., but the "subject," who was very right-handed, had broken her right wrist a year ago, thus no doubt lowering the speed of this hand. Another teacher was noted as 80 per cent., and she had always considered herself ambidextrous ; she had, however, quite lately injured her left thumb, which would account for a decreased speed with her left hand. A good many experiments have been made with other adults, and in no case, except those given below, was the percentage less than 29 per cent. for the right-handed.

(ii) Line test : percentages for left-handed adults, and for doubtful cases.—

(*a*) Known to be left-handed, but always write with right hand : 21 ; 13 ; 7 ; 6 ; 5 ; 4 ; 1 ; 0 (two cases) ; 2 (–).

(*b*) Doubtful cases :—

Twenty-three per cent. Thinks she is right-handed, but in dressmaking always cuts on the left side with left hand ; draws equally well with left or right hand ; always ties left-handedly.

Fifteen per cent. Does many things left-handedly ; has always thought herself ambidextrous.

Five per cent. Did not know he was left-handed ; always deals cards left-handedly ; throws with left or right equally well.

Five per cent. Not known to be left-handed, but in boxing equally strong with left or right ; kicks equally well with either foot ; grip dynamometer : right hand 107 kilos., left 105 kilos.

Two per cent. Carves and draws equally well with left or right ; left arm (ulna) an inch longer than right ; had once suffered from injury to right hemisphere with temporary loss of use of left hand and leg, but with no speech defects.

The average number of lines in thirty-six cases was for the right hand 68·5, for the left 56·5. The greatest number for right hand 88, for left 87 ; the least number for right 39, for left 21.

(*g*) *Grip test.*—This is a well-known test to measure by means of a dynamometer the strength of grip of the right and of the left hand; a great many observations have been recorded by various authorities. The norms are given for boys and for girls at different ages in the "Manual of Mental and Physical Tests," by G. M. Whipple. The index of right-handedness, i.e., the percentage of strength of the left hand compared with that of the right, was found to range, for an ordinary group of school children, between 91 and 96 per cent. "However, occasionally right-handed children may have an index of strength exceeding 1·00, i.e., grip may be stronger with their left hand. According to Ardlicka, this tendency to have an index contrary to expectation is peculiarly evident in left-handed persons, so that he estimates that nearly one-half of bona-fide left-handed persons may have a stronger grip with the right hand."

The following quotation from the same book is of special interest in connection with the present investigation. "It is often asserted that degenerates tend to be left-handed. Wallin's averages for epileptics show a net preponderance of 0·5 kg. for general average in favour of the left hand, though by no means all his S.'s had an index above 1·00."

It was thought that the grip dynamometer would be very useful in diagnosing left-handedness, but the results were so varying and often so inconsistent with known facts, that little value could be placed on the results obtained by it, unless these were fully supported by other tests. No doubt its inefficiency is due in part to the instrument used, as it is ill adapted to the varying sizes of the children's hands and could not be adjusted. Also there was not sufficient time to allow each child to have as a rule more than one grip with each hand. The dynamometer was taken round to each child, and placed in his hand in the best position and he was told to squeeze as hard as he could. The result was then called out and recorded by the teacher. In this way every one was stimulated to do his best. As a matter of fact every child was eager to make the trial, and wished to try over and over again to improve his record. One curious result was noticed, namely, that in the first trial the two hand grips often approached very closely, not only among mental defectives, but also among normal children and even among adults. After practice the difference between the two hands becomes more marked, but even then it is by no means constant.

Some of the results are recorded in the tables of tests at different schools, but the results must be regarded with a good deal of suspicion.

(*h*) *Other tests.*—Many of the following tests have been tried, but

not very extensively. Generally they gave results agreeing with those obtained by the other tests, but only single individuals and those as a rule who were left-handed (or doubtful) were experimented with.

(i) Placing counters in a box one by one, two children being raced to see who could do it the quicker. As a rule it was done by the dominant hand.

(ii) Tying a knot in a piece of string: this was found to give very contradictory results, especially by those who had done "string" work at school.

(iii) Hammering nails and sawing wood, among the elder boys: if done left-handedly the boy was nearly always left-handed.

Taking the tests as a whole, the most unsatisfactory in the case of children in mental defective schools were those quantitative in character, and requiring attention and concentration. The tests which gave the most consistent and reliable results were those in which actions that had apparently become fixed by habit at some time in their early childhood were tested.

§ 2.—*Method of Testing Classes.*

(*a*) *In junior mental defective schools in infant schools and in Standards I and II of the ordinary schools.*—A list of children with their ages is given to the teacher, and each child is tested with the duster as has already been described. The teacher notes those who rub and throw with the left hand. Only those who rub with the left hand are at this stage tested in throwing. Each child is then tested with the scissors, and those who cut with the left are also tested with the right hand, the results being noted by the teacher. Any child found to cut with the left hand is also tested in throwing, if he has not been previously tested, and the result entered as left or right. Sometimes the dynamometer grip test is then tried; at other times the "line" test. Finally those indicated as left-handed in anything are raced in "picking up" and in "winding."

The teachers are then, and not till then, asked to give the names of those who write with the left hand, who used to write or who wished to write with the left hand; also those who draw with the left hand. If any of these have been missed they are tested in throwing, &c. Often in the case of very young children they are asked to draw something on their millboards, and a note made of the hand used.

Some examples of classes tested in this way are given in the tables.

(*b*) *In elder boys' and girls' schools.*—A list of children with their ages is made as in the infant schools, and the teacher records the results. The first test is the "throwing" test, which is followed by the "line" test. In Standards V, VI, and VII this latter test takes very little time, as after the lines have been drawn the children are asked to count the number made by each hand and enter it on the slip. The teacher then calls out each name, and the children read out the number for the right and for the left hand. The slips are collected for scrutiny and verification; they are especially examined in those cases where the number of lines approach each other, or where the lines made by the left hand are more numerous than those made by the right. Each child is then tested with the scissors. Those who appear to have indications of left-handedness are further tested with the "picking up," and "winding" tests. Sometimes the dynamometer is used in addition to, or in place of, the "line" test. Information as to the hand used in writing is then obtained. Examples of the results recorded in classes for older children are given in the tables.

The tests which have just been described appear to be more suitable for children in mental defective schools than quantitative tests (e.g., as tapping, &c.),[1] as the latter involve attention and concentration, both of which are difficult to obtain from these children. In addition, much time and care is required in using them, and the children's skilfulness is only proved in one or two special kinds of work. The qualitative tests, on the other hand, reveal much of the past history of the child, and give evidence of habits, natural or acquired. As a matter of fact, very few were missed who were known to be left-handed, or perhaps it would be better to say, who were known to write or to have written at one time or another with the left hand. In many cases left-handed children were discovered who were not known by their teachers to be left-handed, but who on inquiry were proved to have been left-handed at school or at home. One thing also became clear, namely, that there were all grades of left-handedness. There were some indeed who did everything left-handedly; others who did most things left-handedly, and again others who were right-handed in most of their activities, but had some clearly marked left-handed habits.

At first sight these conclusions may seem to depreciate the value of the tests, but it will be found that the results of the tests are by

[1] "Shot tube" test, that is, dropping steel balls into a vertical glass tube, and comparing times taken by each hand. "Pegboard," in which pegs are fitted into holes, and the times taken by each hand compared.

TABLE I.—SCHOOL, MENTAL DEFECTIVE: CLASS 1.

Names	Ages		Rub	Throw	Cut	Pick up	Wind	Write	
Boys—	Yrs.	mths.							
1	12	0	..	..	..	..	..	..	..
2	14	3	L.	L.	L.R.	R.	L.	R.	Always cuts with left at school
3	10	1	..	..	..	..	..	..	..
4	12	1	..	..	..	..	..	..	..
5	9	9	L.	R.	L.	R.	L.	R.(L.)	..
6	11	3	..	..	..	..	..	..	..
7	11	10	L.	L.	L.R.	L.	L.	L.	..
8	12	0	..	..	..	..	..	..	..
9	11	5	..	..	..	..	..	..	..
10	11	4	..	..	..	..	..	..	..
11	11	4	..	..	..	..	..	..	..
12	12	6	..	..	..	..	..	..	..
13	11	5	..	..	..	..	..	..	..
14	11	2	..	..	..	..	..	..	..
15	11	0	..	..	..	..	..	..	..
16	9	5	L.	L.	L.	R.	L.	R.(L.)	..
17	11	4	..	..	..	..	..	..	..
Girls—									
1	12	3	L.	L.	L.R.	L.	L.	R.(L.)	..
2	14	0	..	..	..	..	..	..	..
3	9	5	..	..	..	..	..	..	..
4	9	5	L.	L.	0*	L.	?	R.(L.)	..
5	9	9	..	..	..	..	..	..	..
6	9	3	L.	R.	0*	L.	?	R.(L.)	..

(1) All were tested in rubbing and in cutting, only those indicated as left-handed were further tested in "picking up" and "winding."

(2) All were tested in left-hand writing; no mirror writers.

(3) Under "write" are given the hand habitually used in writing: (L.) indicates that these children were known at the school to have written, or to have wished to write, left-handedly at some period of their school life.

(4) Under "wind" ? indicates that both hands were used at the same time, and that it was therefore impossible to decide which was the dominant hand.

(5) Under "cut" L. = could cut with left *only*; L.R. = could cut equally well with left and right; L.(R.) could cut with left and not at all well with right.

(6) * = not tested in this, e.g., right-hand cutting.

TABLE II.—SCHOOL, MENTAL DEFECTIVE: CLASS 2.

Names	Ages		Rub	Throw	Cut	Pick up	Wind	Write	
Boys—	Yrs.	mths.							
1	8	11	..	..	..	..	..	..	..
2	10	5	..	..	..	..	..	..	..
3	9	7	..	..	..	..	..	..	..
4	9	10	..	..	..	..	..	..	..
5	10	6	L.	L.	L.	L.	L.	L.R.	Speech indistinct; *has* written mirror
6	8	8	L.	L.	L.R.	L.	L.	L.	Decided epicanthus; *has* written mirror
7	7	11	..	..	..	..	..	..	..
8	7	10	..	..	..	..	..	..	..
Girls—									
1	10	8	L.	L.	L.	L.	L.	L.	Defect of articulation; *has* written mirror
2	9	7	..	..	..	..	..	..	..
3	8	3	L.	L.	R.	L.	R.	L.	Very backward and babyish
4	12	7	..	..	..	..	..	..	..
5	8	4	..	..	..	..	..	..	..
6	10	7	..	..	..	..	..	..	..

no means unimportant, when the reason for the larger number of left-handed children in mental defective schools comes under consideration.

In deciding whether a child should be classed as left-handed—in estimating percentages—the following tests as a rule were taken as sufficient :—

(1) Persistent rubbing and throwing with the left hand, especially among very young children ; these nearly always also wrote with the left hand.

(2) Throwing and cutting with the left hand.

(3) Writing or having persistently written at one time with the left hand.

TABLE III.—SCHOOL—MENTAL DEFECTIVE, ELDER BOYS : CLASS 2.

Names	Ages		Throw	Scissors	Dynam.		Pick up	Wind	Write	
					L.	R.				
Boys—	Yrs.	mths.								
1	14	4	..	..	40	49	..	..	..	..
2	13	1	..	..	66	75	..	..	..	Best boy but difficult to control
3	14	1	..	..	50	51	..	..	..	..
4	14	3	..	..	10	13	..	..	..	Fairly intelligent. Attending hospital
5	14	7	L.	L.R.	47	54	L.	L.	R.(L.)	Fairly intelligent
6	14	11	..	..	41	28	..	..	..	..
7	14	2	..	..	43	47	..	..	..	..
8	13	9	L.	L.	45	36	L.	L.	L.	Fairly intelligent
9	13	9	..	..	44	40	..	..	..	..
10	13	10	..	..	48	40	..	..	..	..
11	15	1	..	..	35	35	..	..	..	..
12	13	8	..	..	31	29	..	..	..	..
13	13	7	..	..	18	20	..	..	..	..
14	15	11	..	..	30	31	..	..	..	..
15	14	0	..	..	34	39	..	..	..	..
16	13	4	..	..	40	38	..	..	..	..
17	14	3	..	..	33	33	..	..	..	..
18	14	5	..	..	24	30	..	..	..	..
19	16	8	..	..	49	55	..	..	..	..
20	15	3	R.	O.*	38	38	R.	?	R.	*Mirror* writer ; very slow. Chorea at one time

Note.—All tested with scissors, throwing and dynamometer ; in the other tests only those indicated were tested. All tested in left-hand writing.

Children who could cut with the left hand, but displayed no other left-handed activities, were classed as right-handed. In many cases, however, further inquiries were made into their past history. Children who merely "pick up" or "wind" or do both, were not included among the left-handed. The dynamometer and "line" tests were only used as confirmatory tests. In the great majority of

cases, especially in mental defective schools, there was little or no difficulty in coming to a decision. In doubtful cases the children were classified as right-handed.

When it appeared that the percentage of left-handed children in mental defective schools was very much higher than had been found by various authorities among normal children and adults (4 to 6 per cent., and sometimes higher), it became necessary to apply the same tests to the children in the ordinary elementary schools. The same methods were used—one set of tests for children up to about 9, and the other set for older children. As will be seen by the results, the percentage was very much lower than that in the special schools, and somewhat higher than had been estimated by the majority of authorities.

Chapter III.—Results among Normal and Mentally Defective Children.

Among normal people, Ogle found $4\frac{1}{2}$ per cent. left-handed in 2,000 cases; G. M. Gould gives 6 per cent.; Franklin Jones, 4 per cent.; H. Macnaughton Jones says: "Ghent University had 22 per cent. left-handed (100 cases), but generally the left-handed are estimated at 1 per cent. to 2 per cent."[1] Malgaigne (1859) 8 per cent. to 10 per cent.; Hecht and Langstein (1900), even 12 per cent.; Masini (1907), 10 per cent.; Songues (1,080 cases), 7 per cent., left-handed and ambidextrous; Moutier, at least 10 per cent.

Ballard, in "Sinistrality and Speech" (1912), gives 4·1 per cent. as the result of data obtained from the teachers in reference to children in elementary schools in London. The number of children dealt with was 13,189, and the ages between 4 and 14 years. This percentage is considerably lower than that (7·3) found in elementary schools in Middlesex and London during the present investigation; the number, however, actually tested was only 3,298, and the ages between 4 and 14 years. In his own personal inquiry, however, Ballard reduced the percentage to 2·8; 11,393 cases, and ages between 8 and 14 years. This reduction is remarkable. It seems possible that the omission of the infant schools may have been the cause of the difference in percentages. It is possible that left-handed children may have been over-

[1] Josefa Ioteyko, late of Brussels University, states (1917) that there are a considerable number of left-handed in Belgium much above the general estimate of 2 to 3 per cent. [10]

looked in the senior schools, owing in part to a forced change in writing.[1]

To discover whether anything of the kind had occurred in the present inquiry, the percentage of left-handed in Infant schools and in Standard I was compared with that in Standard II and upwards, with the following results :—

	Number tested	Number of left-handed	Percentage
Senior schools, Standards II to VII	934 ..	77 ..	8·2
Infant schools, and Standard I	910 ..	64 ..	7·0

In the estimates of left-handed people the outstanding feature is their great variety. There can be little doubt that percentages in a subject of this kind are very misleading, and that they are of little value unless large numbers are dealt with. The present inquiry has clearly shown how greatly classes and schools differ from one another; for example, in one class of a school 20 per cent. were found to be left-handed, and in another class in the same school there was none. In one class of a mental defective school 66 per cent. (14 out of 21) were clearly left-handed, whereas in another school of the same kind there were only 1 to 2 per cent. Apart from this, different methods give very different results. In fact left-handedness has always been very difficult to diagnose. Personal interviews are very misleading and inaccurate; children are as a rule very unwilling to acknowledge left-handedness and have frequently forgotten all about it, even in cases in which they still use the left hand as the leading hand in many of their activities. The older the "subject" the more difficult is the diagnosis. As a matter of fact many undoubted left-handed adults are convinced that they are ambidextrous. The question naturally arises as to how we are to define left-handedness. Moutier suggests that a left-handed person is one who habitually does with the left hand a series of professional and usual acts generally done by the right hand in the case of the right-handed, and that the ambidextrous are those who do these acts with either hand indifferently. In the present investigation, a definition of a very similar character was the basis of the diagnosis of left-handedness, although the work of Moutier on aphasia had not been seen until almost the end of these experiments and observations. Although this definition is open to criticism, it has, I think, been fruitful in suggesting experiments and inquiries, which have not been altogether barren of results.

[1] Schäfer (1911) [23], in an investigation by questionnaire, concludes that the percentage of left-handed is higher among boys than girls, and that it diminishes at each age.

Results of tests :—

Schools	Number tested	Number found to be left-handed	Percentage
(1) Elementary schools, ages 4 to 14	3,298	241	7·3
(2) Mental defective schools, ages 8 to 16	4,620	843	18·2

There appears, therefore, to be more than two and a half times as many left-handed children in mentally defective schools as in ordinary schools.

To discover whether there was any great difference in the percentages of left-handed boys and girls in these special schools, 729 cases were taken haphazard with the following result :—

PERCENTAGE OF LEFT-HANDED BOYS AND GIRLS.

	Number tested	Number found to be left-handed	Percentage	Percentage boys and girls
Boys	374	61	16.6	18·7
Girls	355	76	20·7	

These numbers are not very large, but they appear to represent about the proportion generally found. The proportion of girls to boys as found above is therefore as five to four, in other words the left-handed girls are about 25 per cent. more numerous than the boys. J. W. W. Wallin (1915-16) [26] found by questionnaire the percentage of left-handed among boys 3·6 and among girls 2·1. The number of pupils dealt with was 89,057.

Whether the above percentages are reliable or whether they are too high, one thing is clear to anyone visiting both special and ordinary schools, namely, that there is a very much higher percentage of left-handed children in the special schools, no matter what method of estimating the number is used, whether by merely noting those who write and work left-handedly, or by testing to discover in addition those who were at any time left-handed and have been converted into right-handedness in many of their activities, such as writing, throwing, sewing, knitting, &c.

Some very high percentages were found in some of the classes and in whole schools, as is shown by the following percentages of left-handed children in certain classes, and in whole schools (mental defective).

53 per cent. (47) ; 45 per cent. (19) ; 44 per cent. (61) ; 33 per cent. (27) (39) ; 31 per cent. (19) ; 29 per cent. (17). For whole schools : 2 schools over 30 per cent. ; 5 schools 25 to 30 per cent. ; 13 schools 20 to 25 per cent. ; 12 schools 15 to 20 per cent., &c.

The numbers in brackets are the actual numbers tested. In no case was a class specially selected.

In ordinary schools the percentages varied very considerably in different classes. Sometimes the whole school was tested, but generally a few classes were chosen haphazard. The percentages were as a rule between 4 and 10 per cent. In three cases the classes were specially tested owing to the known high percentage, e.g., (i) a backward class gave 15 per cent., whereas three other classes in the same school gave 5 per cent., 3 per cent. and 3·5 per cent. ; (ii) Standard Ia with 22 per cent., two other classes in the same school gave 3 per cent. and 6 per cent. respectively ; (iii) Standard II with 13 per cent., whereas Standard I gave only 2 per cent.

From these results the question naturally arises: Why are there so many more left-handed children in mental defective schools? The answer to this question will be left for the present.

Chapter IV.—Twins and Left-handedness.

In an elementary school two girls (twins) 14 years of age and nearly identical in all their characteristics—school work, appearance, &c.—were found on testing to be, one thoroughly left-handed, the other right-handed. On the next day in a mental defective school, twins—boy and girl—gave the same results, i.e., one left-handed, the other right-handed. Sometime later, it seemed probable that an investigation on the "handedness" of twins might throw some light on the hypothesis suggested later in this paper. There appear to be two kinds of twins —the one being derived by the fertilization of a single ovum, the other from the fertilization of two distinct ova. In the first case the twins are stated to have exactly the same characteristics, and it seemed that it would therefore be possible to decide whether left brainedness was one of these characteristics. As will be seen by the facts given below, it is by no means so simple a question as appears at first sight, as it is quite possible that the two twins may originally have been "identical," but that one may be very unlike the other in some of the characteristics (such as height, mental capacity, &c.) owing to some accidental cause.

Galton [8] in speaking of twins states that "the word twins is a vague expression, which covers two very dissimilar events—the one corresponding to the progeny of animals that usually bear more than one at birth, each of the progeny being derived from a separate ovum, while the other event is due to the development of two germinal spots

in the same ovum. In the latter case they are enveloped in the same membrane, and all such twins are found invariably to be of the same sex."

The following results were obtained from 219 pairs of twins, most of whom were in ordinary elementary schools. They were all tested in the usual way described in this paper.

TWINS.

Sex	No.	Both Right-handed	Both Left-handed	One Right, other Left-handed
Boys ..	59	47	0	12
Girls ..	79	69	1	9
Boy and girl	81	53	3	25 (i.e., 11, boy R. and girl L.; 14, girl R. and boy L.)
Total ..	219	169	4	46

It is remarkable that in the mixed twins (boy and girl) nearly one-third the number consists of the one right-handed and the other left-handed. As to ability, appearance, &c., about 66 per cent. both of boys and of the girls are very much alike, when both are right-handed, and 33 per cent. are very dissimilar.

Some curious facts were noticed in the case of twins, one of whom was left-handed and the other right-handed, facts which are shown in the following table of comparison of the left-handed twin with the right-handed. The notes as to ability and physical defects were obtained from the teachers before the tests were made.

It will be noticed that in eight cases the left-handed twin is mentally defective and the right-handed is in an ordinary elementary school. Hitherto no case has been reported of the right-handed twin being in a mental defective school and the left-handed in an ordinary school. Not infrequently both right-handed twins are in a mental defective school. In twelve cases the left-handed twin is very nervous, much shorter, or more retarded compared with the right-handed.

It is possible with some of the twins (left and right-handed)—

(1) Especially where both are quite normal, that the left-handedness of one may be due to some accident as given in the note (p. 335), or

(2) Where the right-handed is much retarded, that they were originally both left-handed, e.g., in case of chorea, &c.

Comparison of Twins, one of whom is Left-handed, and the other Right-handed.

Both Boys.

	The left-handed twin	The right-handed twin
1	Very nervous	Normal
2	Very nervous, cries, shorter, less intelligent	,,
3	Normal	Rather retarded (a brother is left-handed)
4	Very short, in Standard IV	Very tall; in Standard IV
5	Left eyelid droops; in Standard IV ..	In Standard V
6	Healthy; average ability	Rather dull; adenoids and nasal catarrh
7	In mental defective school; defective speech	Died at 9 years of age; ordinary school
8	In mental defective school; hesitates in speech	In Standard IV
9	In mental defective school; 15 years old ..	Was in mental defective school; allowed to leave at 14 as much progress made

Both Girls.

1	Normal (very alike)	Normal
2	Dull	Dull; chorea
3	Much shorter and more babyish	Ordinary
4	In mental defective school; 8 inches shorter than sister, and much less developed	In mental defective school

Boy and Girl.

	Girl left-handed	Boy right-handed
1	Very slow; "peculiar speech"; improving, now writes right-handed	Normal
2	Chorea	,,
3	In a mental defective school	,,
4	Examined for mental defective school; on probation; very small	Normal, small
5	Below average intelligence	Dull
6	Very bright	Comparatively dull
7	Normal	Normal
8	Good ability; shorter, delicate; glandular abscess in neck and leg	Better ability than sister
9	Standard V	Standard VII; suffers from heart
10	Backward; lungs affected	Very bright

	Boy left-handed	Girl right-handed
1	In mental defective school	In mental defective school
2	,, ,, ,, ...	Below normal
3	,, ,, ,, ...	Fairly intelligent
4	Above normal	Above normal
5	Average ability	Below average ability
6	Not so bright as sister	The brighter
7	Normal; left eye squint	Normal
8	Standard VII	Standard V
9	In mental defective school; very defective speech	Standard V
10	In mental defective school	Standard VI; delicate
11	Much shorter than sister in infant school ..	Under-sized; in infant school

Note—In the statistics given are included all the twins in elementary and mental defective schools, who have been tested. Among the twins classified as both right-handed were:—

(1) Both girls, in an infant school, one did most things left-handedly, but she was right-handed until she broke her arm three months previously.

(2) Both girls, aged 13½ years: one left-handed in everything except writing. She was right-handed until she burnt the palm of her right hand three years ago; she has only just recovered, the palm is very badly scarred.

CHAPTER V.—MIRROR WRITING.

As has already been mentioned, many children in mental defective schools were found to write mirror fashion when writing with their left hand, and as this kind of writing was prevalent in these schools, various experiments and inquiries were made in reference to the matter. The ages of the children who wrote mirror fashion varied greatly—from about 10 to 16 years; younger children could not as a rule be tested, as they were not able to write their names. With one or two exceptions the mirror writers were right-hand writers, but it was remarkable that the left-hand mirror writing of most of these children was much better than their ordinary writing with the right hand.

As a matter of fact, the mirror writing in the case of 56 per cent. was very good indeed, of 29 per cent. good, and 7·5 per cent. fair, and of 7·5 per cent. bad.

57 per cent. started at the right-hand edge of the paper, 13 per cent. in the middle, and 30 per cent. towards, or not far from, the left margin—the usual place of beginning in ordinary writing. At first, there seemed to be a close connection between the act of mirror writing and the starting position; e.g., one began correctly, but after writing the first letter, which was at the extreme edge of the paper on the right, finished mirror fashion. Another began at the extreme right edge and wrote each letter correctly, but from right to left, i.e., backwards.

Some began mirror fashion, and apparently noticed that something was wrong, and then tried to correct themselves; in some cases successfully, but in others without success, even after trying two or three times. Their hand appeared to be doing exactly the opposite to what they wished. The majority were unable to read what they had written, but many could read an easy word, such as "cat" written mirror fashion. As a rule, the writing was done very rapidly and without hesitation, even by the very "low grade" feeble minded. But there were other cases where it was evident that the child was following carefully with the eye every letter, and it seemed certain that the letters were being formed in exact accordance with an optical mental image formed at the time. Generally the writing in these cases was slow, but it was well done. How remarkable this mirror writing is can be easily understood when it is explained that intelligent and capable teachers, who have not practised mirror writing, find great difficulty in writing in a "running hand" a capital (not printed) G or L mirror fashion, and

yet these very mentally defective children could do it without any difficulty or effort.

A few print their names (mirror fashion), but as a rule the writing looked at in a mirror is very similar to the ordinary writing of the child. In one or two cases the name is printed with the right, and written in a "running hand" mirror-fashion with the left; these latter cases are in schools which have lately taken up "print writing."

One boy, a left-hand writer, wrote mirror fashion with his right hand. There were there cases of the same kind, and also mirror writing with the left hand by children who habitually write with the same hand.

Various experiments were made with the children who wrote mirror fashion, e.g., they were asked to write "cat," "10," "25," &c., with their left hand and then with their right. In most cases, the left-hand writing was mirror fashion, and the right in the ordinary and correct way. Sometimes the right-hand writing was done first, but the results were the same, namely, mirror writing with the left.

Again, others were asked to write "cat," "dog," "45," with the right hand, and then to copy them underneath with the left; the result was the same. The writing was done as a rule without any hesitation, and with rapidity. A word, e.g., "cash," was written mirror fashion, and some of these children were asked to copy it underneath with their right hand; they generally copied it correctly, i.e., not mirror fashion; on being asked to copy what they had just written, but to do it with the left hand, they wrote it mirror fashion. In one experiment the word "illustrate" was written mirror fashion in a running hand by the experimenter. The child could not read it, but when asked to copy it with her right hand she immediately wrote the word correctly (not mirror fashion) in every detail.

There are a few letters and figures which are frequently written mirror fashion by young children, especially s, d, b, 4, 6, and 9, but this writing is undoubtedly different from the mirror writing under discussion, and may be due to the lack of accurate observation on the part of a child, and never seems to extend beyond single letters and figures; but of course correct writing with one hand and mirror writing with the other is due to some other cause.

At one school, a little girl (D. E.) copied in print a whole page from a book mirror fashion, with the exception of "a" and "e": she had never done this before, and she had not done it since. She was tested in the usual way with others in the class; she "rubbed," "threw," and "wound"

with her left hand, but "picked up" with her right, and could only "cut" with her right; her "grip" was 2 kilos with each hand; she has a "peculiarity" in her speech. On thinking the matter over, it seemed probable that she had done the copy with her left hand, so at the next school, where two mirror writers were found in one class, they were both asked to copy out a page from a book, and to do it with the left hand. They were both right-hand writers, but they made the copy rapidly—one was well and correctly written, the other was well written, but every letter and word was written mirror fashion. This girl (E. A.) aged 14, is hydrocephalous and very defective: she "threw," "picked up," and "wound" with her left, could "cut" with either hand, her "grip" was 12 kilos with each hand,[1] but she always "rubbed" with her right hand. At a subsequent visit to the first school, the little girl (D. E.) was asked to copy some lines from a book with her left hand, and to begin at the right-hand corner. She immediately wrote her name mirror fashion, and then wrote a whole line in a similar way. When she reached the end of the line, i.e., at the left-hand margin of the paper, she asked whether she should go back the other way, that is, starting just under where she had finished. She then wrote this line quite correctly, not mirror fashion. The same experiment was tried with her right hand, only she was asked to begin in the middle of a line. She wrote correctly until she reached the end of the line, and was then asked to go on writing from the position just under where she had finished; without any hesitation she wrote this line mirror fashion with her right hand. This little girl was 9 years 2 months old. The doctor reported when she was examined (aged 8 years 8 months): "Talked at 5 years; walked at 2 years. Defective articulation: left-handed." Until three months ago she used to write with her left hand, but not mirror fashion. After a few months writing with her right hand, she writes mirror fashion, when she reverts to her left hand. Further, she now writes mirror fashion with her right hand when she is started at the right edge of the paper. It would therefore appear that in her case at all events the change in writing from one hand to the other may have had something to do with the peculiar kind of writing. As will be shown later (p. 342) a very high percentage of children, who have at one time written with the left hand but now write with the right, write mirror fashion when asked to write their names with the left hand. It is important to note that this girl did not write mirror fashion when she first began to write with her right hand, but that it was only after she had practised writing with her left and also with her right hand. The importance of this fact will be understood when the hypothesis suggested by Baldwin is considered (p. 353).

Subsequently it was found, especially in the case of children, who at one time had written mirror fashion, and of others who sometimes write with the left hand, and at other times with the right hand, that they can be made to write mirror fashion by merely asking them to

[1] Normal grip for a girl of this age: right 24·79, left 22·92 kilos.

begin at the extreme right margin of the paper. The probability of such a result only suggested itself quite lately, so only a few experiments have been made. Teachers were asked for the names of children who sometimes write with the left and sometimes with the right hand. The following results were obtained from a few children selected in this way.

H. N., boy, aged 8. The doctor reports: "Speech defective. Father epileptic (?), and cannot read or write." The boy when asked to write his Christian name with his left hand beginning at the extreme right edge of the paper, wrote in mirror fashion without any hesitation. He wrote his name correctly with the same hand, when asked to begin at the left margin of the paper. With his right hand he did exactly the same, i.e., mirror fashion at the right edge, and correctly at the left. There was, however, one peculiarity in all the four written names, namely, the correct writing (not mirror fashion) of the final "y" in his name (Henry). At another school very similar results were obtained with a left and right hand writer. This boy with his left hand wrote his name completely mirror fashion without hesitation when started at the right margin, also "cat," but "25" correctly; with his right hand, however, under the same conditions, he hesitated and seemed to find some difficulty in beginning, but finally wrote his name completely mirror fashion. At the left margin, he wrote his name correctly with either hand. Other experiments were made with left-handed children, but some of them could not be induced to write mirror fashion with either hand, when asked to begin at the extreme right margin, as they insisted on edging off to the left and then wrote their names in very small letters, so as to get them into the only available space. Other mirror writers (with left hand), many of whom wrote mirror fashion wherever they were started, could not be induced to write mirror fashion with the right hand under any circumstances.

Another interesting case was that of a boy (H. W.), left-handed in writing and in nearly everything else, except throwing, who wrote all letters and numbers correctly with his left hand, but wrote them mirror fashion with his right.

A curious case was met with at another school. A little girl (L. C.) wrote a word correctly with her left and mirror fashion with her right: she was asked to write another word first with her right—she wrote it correctly; but she wrote it immediately afterwards mirror fashion with her left hand. She is 12 years 9 months old: throws left, cuts left only, winds left and writes left. The doctor reports: "Infantile paralysis, right hemiplegia, used to write mirror fashion with her left hand; gave it up in 1916 (i.e., about 8 years of age), but reverts to it at times in 1917." At a second visit she printed her name completely mirror fashion on the blackboard with her right hand when asked to begin at the right-hand corner.

Early in the investigation it was noticed that there was a much higher percentage of mirror writers among children, who, although

they wrote with the right hand, were evidently left-handed in many others of their activities. This fact has been noticed by previous investigators. Ballard (1916), referring to mirror writing, says: "With rare exceptions it is done by left-handed people only, and with the left hand only. . . . The subject does not at first recognize the word as he writes it down; nor can he without some practice read it. . . ." [4].

A. L. Beeley (Instructor in Psychology, Utah University), after an investigation in schools of Chicago in 1918, came to a similar conclusion, namely, that mirror writing is "a characteristic of extreme left handedness," and "not necessarily of mental deficiency." Ireland [11], 1881, connected mirror writing with left-handedness.

As mirror writing appeared to be associated with left-handedness, many left-handed adults were asked to write their names with the left hand, but they always did it correctly. In one case, however, it was noticed that the pencil seemed instinctively to have been placed at the right-hand edge of the paper, and then withdrawn and the writing begun at the usual place—left margin—without the "subject" paying much attention to what she had done. An experiment was next made with a left-handed teacher, a capable and intelligent head-mistress. She was asked to write her name with her eyes shut; she did it rapidly with her right hand, and quite as well as with her eyes open. She was then asked to close her eyes, and write her name with her left hand; she did so immediately but mirror fashion. When she looked at it, she could not make out what she had written, and said, "What is the matter with me?" When the paper was held up to the light—the reverse side—an excellent copy of her name in her ordinary writing was seen. She explained that she was quite sure she was writing it in the ordinary way.

Similar experiments with a few other left-handed teachers were made, and in one case with exactly the same result. In fact the latter, a capable and intelligent assistant teacher, made nearly the same remarks as the first. In still another case, although the teacher wrote her name correctly with her left hand, she stated that while writing it she thought at times she was writing it "backwards." A few left-handed adults who write with the right hand were found to be able to write mirror fashion without any difficulty with the left hand, although they had not made the attempt before. Others—right-handed or left-handed—have to concentrate their whole attention on what they are attempting, and even then make mistakes.

McHattie (1913) [20] describes some experiments he made in London elementary schools in connection with mirror writing. He found in the left-hand writing of 1,240 children, ages 7 to 14 years, that there were only seven cases (0·48 per cent.) of mirror writing. One was a girl, she was not included in the percentage, whose right hand had been partially disabled by infantile paralysis, and could not write with this hand. The percentage was about twice as high with the boys as with the girls.

In the present investigation 829 children were tested in left-hand writing in the ordinary elementary schools (infant and senior departments), and only four (0·48 per cent.) were found to write mirror fashion. The total numbers in both cases are small, but they agree very closely, and it appears likely that the percentage (0·4 to 0·5) is not far wrong. A. L. Beeley [6], in elementary schools in Chicago in 1918, found in 103 schools with a school population of 106,356, forty-two mirror writers—i.e., about 0·04 per cent. In his investigation, however, he did not test the left-hand writing of every child, but only asked for information from the teachers concerning all children who had been observed to write mirror fashion at any time in their lives. There was about the same percentage of mirror writers among the boys as among the girls.

The percentage of mirror writers in mental defective schools in London and Middlesex is given below:—

Number of children treated	Number of mirror writers	Percentage
1,350	109	8·0

These numbers include both boys and girls of varying ages from 8 to 16 years, generally about 8 years of age and upwards, as only those who could write their names were tested. Mirror writing of isolated letters and figures—such as 3, b, d, 4, 6, 9—is not included in the above percentage. It is evident, therefore, that mirror writing is very much more prevalent among the feeble-minded than among normal children; the percentage is in fact about seventeen times as great.[1]

A still more striking result is obtained from the calculation of the percentage of mirror writers among left-handed children.

The following statistics have been carefully made out in the case of mirror writers, who had all been tested in "handedness."

[1] In one school thirty-one were tested, and there were ten mirror writers—a most remarkable result. It seemed possible that the mirror writing might have been due to some special methods of instruction in writing, drawing, physical exercises, &c., but after careful investigation it was evident that the methods used were the same as those used in other special schools.

MIRROR WRITERS AMONG LEFT-HANDED CHILDREN.

Sex	Number tested in left-handed writing	Number found to be left-handed	Number of mirror writers among left-handed	Percentage of mirror writers among left-handed
Boys ..	374	61	11	18
Girls ..	355	76	30	40
Boys and girls	729	137	41	30[1]

That is to say, 30 per cent. of the left-handed children were mirror writers, and in the case of girls the percentage reached the astonishing number of 40 per cent.

Further the mirror writers were nearly always left-handed children, who wrote with the right hand. These percentages were further analysed with the following results: 40 per cent.[2] of these left-handed boys habitually write with the left hand, and 60 per cent.[3] with the right hand.

Of these left-handed boys who write with the right hand 32 per cent. wrote mirror fashion with the left.

Curiously enough the same percentage (i.e., 40) of girls write habitually with the left and 60 per cent. with the right hand.

Of these left-handed girls who write with the right hand, 62 per cent. wrote mirror fashion with the left. The majority of mirror writers have, therefore, at one time or another written with the left hand; and it would seem that, this being the case, they would be less likely to write mirror fashion than children who had never co-ordinated their left-hand movements to the usual forms of letters and words.

It is therefore evident that mirror writing is very closely associated

[1] The percentage of right-handed children (i.e., children who gave little or no indication of left-handedness in the tests) who wrote mirror fashion with the left hand, was of boys 1·6 per cent. and of girls 2·8 per cent.

[2] To see whether this result gives a fair estimate, a larger number was taken: of 251 found left-handed 106 habitually write with left = 42 per cent.; 51 are known to have written left = 20 per cent.; 94 are not known to have written left = 38 per cent.

[3] There is a peculiar coincidence in connection with the children in mental defective schools who habitually write with the left hand. As has already been stated (p. 332) 18·2 per cent. of the children in mental defective schools were found by the various tests to be left-handed and, as it is estimated above, 40 per cent. of these are left-hand writers: it therefore follows that 7·2 per cent. of the children in mental defective schools write with their left hand—a number almost identical with percentage of left-handed children in ordinary elementary schools—namely, 7·3 per cent. (see p. 332). It seems, therefore, not unlikely that many of these (7·2 per cent.) may be the "naturally" left-handed children—children whom it has not been found possible to change into right-hand writers.

with mental deficiency,[1] and also with such left-handedness as has been brought to light by the tests already described. It is also clear that mirror writing is not itself due to mental deficiency alone, as only 8 per cent. of the feeble-minded write mirror fashion, nor is it due to extreme left-handedness alone, as very few left-handed children in ordinary and in physical defective schools write mirror fashion. Further, it does not seem impossible that the same cause may in some cases have brought about left-handedness and also mirror writing; that in fact something may have affected the dominant (in these cases the left) hemisphere. It may have been a lesion, causing among other things slight hemiplegia, or it may have been due to defective development of the dominant hemisphere. It would then follow that such left-handedness differed from what is usually termed "natural" left-handedness.

It is very necessary, therefore, at this stage to consider more carefully what is meant by "left-handedness," and also its various forms and their causes.

There has been much discussion and controversy, since Broca's investigations on aphasia, as to whether there is a dominant side of the brain, and whether its dominance is indicated by left or right-handedness. The origin of the controversy arose from observations of a large number of cases of aphasia associated with right hemiplegia, especially paralysis of the right hand, and of a much fewer number with left hemiplegia. Whether the motor speech centre is located in the third frontal convolution, as suggested by Broca, or in the lenticular zone as contended by Marie, or elsewhere, it is unnecessary to discuss in this paper. But it is agreed upon by all that in the majority of cases it is located in the left hemisphere, and further it appears to be held by most authorities at the present day that not only is the speech centre, but that also other important language centres are located in the dominant hemisphere: for the right-handed in the left hemisphere, and for the left-handed in the right.

Moutier in 1906, however, contended that the percentage of cases of left hemiplegia associated with aphasia was much less than ought to have been the case, if this theory was true; and in addition he cites six cases where the aphasia was associated with hemiplegia of a kind opposite to the known handedness of the patient. Moutier's

[1] A. L. Beeley [6], as already mentioned, considers that mirror writing is not necessarily a characteristic of mental deficiency. This is no doubt true, but it is certainly more prevalent among feeble-minded than among normal children.

estimates, however, of the percentage (10-12 per cent.) of left-handed are much higher than those usually recorded. In the cases he has collected (1864-1907) and given in his book, the percentage of cases of aphasia associated with left hemiplegia appears to be about 8·0 per cent. (total cases 347, 318 aphasia with right hemiplegia and 29 with left hemiplegia). He also quotes the following results as given by Hammond (1871): 17 cases of aphasia with left hemiplegia (i.e., 6·4 per cent.) and 247 cases of aphasia with right hemiplegia. In respect also of eighty-two autopsies, in two cases (i.e., 2·4 per cent.) the lesion was located in the right hemisphere. Taking the above 676 cases in all noted, of these there were forty-eight in which the right hemisphere appeared to have been affected. This gives a percentage of 7·1 per cent., a number almost identical with 7·3 per cent., the percentage of left-handed children found in elementary schools during the present investigation. This latter percentage (7·3), as will be seen later, is probably too high, when left-handedness is considered from the point of view of a dominant hemisphere. It must be again emphasized that the above number (676) is far too small a one on which to base a percentage in a matter in which there is such a great variability, as has been shown to be the case in left-handedness.

Again, in reference to question of dominance of one hemisphere, Laurence (1869) asks the pertinent question: Why does atheroma (degeneracy of the walls of the arteries) more often attack the vessels of the left hemisphere?

If lesions among adults—apart from tumours, injury, &c.—are found more frequently in the left hemisphere, and in a proportion even approximating to the percentage of the right-handed, then such a fact would seem to be a strong argument in support of the theory of a dominant hemisphere, and of the belief that the dominant side is the one that is more likely to give way under strain in certain diseases of the brain; that in fact the hemisphere that experiences the greatest strain will be the one to break down first.

If there is a dominant hemisphere, and if this dominance is revealed by "handedness," then those with a dominant left hemisphere will be right-handed, and those with a dominant right hemisphere will be left-handed. There will, however, be some who have been driven to the use of the non-dominant side in all or only in some of their activities by various causes, such as are given below.

Various Causes of Changes of Handedness.

(i) *Very simple causes and requiring no explanation.*—(*a*) Loss of right arm, hand, &c., even the loss of a right thumb, appears to have been sufficient to make a child completely left-handed, e.g., a child without right thumb gave the following results, when tested: rubs, throws und writes with left hand, and of course can only use her left hand in cutting with the scissors. Again, a head teacher stated that she is very left-handed, but writes with her right hand; and when tested she gave every evidence of left-handedness. Subsequently she was told by her mother that she was not originally left-handed, but that at 3 years of age she had very seriously injured her right thumb-nail, an injury which prevented her using her right hand for nearly three years. (*b*) Broken right arm: girl, age about 7 years, broke her arm a year ago, attended three months with her arm in a sling. Tested: could cut with left and right, and could write equally well with either hand; in everything else was right-handed. (*c*) Injured right hand: girl, Standard I: writes with left hand, as she had once injured her right hand.

(ii) *Injuries to the nervous or muscular systems.*—Various forms of hemiplegias; in some cases the affected hand cannot be used at all, in others to a limited extent, and in still others the effect is apparently only transitory, and perhaps often unnoticed. All degrees seem to be found in mental and especially in physical defective schools. It would seem that in some cases the affected hand has continued to be used in writing throughout, but that the unaffected hand has been resorted to in many other activities. A very interesting case was met with in one of the mental defective schools, the history of which is given as fully as it could be determined. Like most of the cases met in these schools it is very difficult to obtain accurate information, especially from parents, with reference to dates.

H. H., girl, born April 13, 1906 (even this date was uncertain; it was given at one school as April 13, 1905, and might have been April 13, 1907). Walked at 1 year 10 months, talked at $1\frac{1}{2}$ years; entered an infant school in 1910 about 4 years of age. The head teacher remembered the girl as being normal and bright, and rather "chubby," and not left-handed. From there she went to the seaside, and then reappeared at the girls' school (senior department of the infants' school) on September 22, 1914, i.e., at $8\frac{1}{2}$ years of age, and stayed until October 1, 1914. The head teacher remembered her as not being at all mentally defective, but troublesome and a bad influence on the younger children; and for this reason she was placed among older girls, Standard V(a); she was

in this school only eight days. She next entered another girls' school in the neighbourhood on November 9, 1914, and remained there until November, 1915. She was placed in Standard I. The attendance registers showed that she had made very regular attendances until May 21, 1915. After that date no attendances are recorded until September 21, 1915, and after that the attendances were very irregular until November, 1915. On May 21, 1915, she was apparently 9 years old.

Her mother states that the girl fell from a chair on her head, when she was 8 years old (9 years?) and was insensible from 6 p.m. to 10 p.m.; that this fall produced concussion of the brain; that "there was a bruise on the left temple," and that "the left leg has since had a lot of veins outstanding"; that "she was silly for about four months suffering from loss of memory." It would, therefore, seem probable that the accident occurred on May 21, 1915, as everything points to this date except the assumed age. The head teacher remembered her as being not very bright (as she remained in Standard I for a year), and she thought there had been some accident. The teachers did not think she was left-handed. In December, 1915, i.e., at 9 years and 8 months, she was examined by the medical officer and sent to the mental defective school on January 11, 1916.

At the original medical examination the doctor reports: "Father and grandfather suffer from rheumatism. Child, giddy and hysterical; fits not epileptic (major); an accident nine months ago: concussion of brain by fall, unconscious four hours. Reading, only little words; writing, scarcely legible; drawing, very little." The head teacher of the mental defective school reports, that the girl often makes grimaces and complains of headaches, and her hands are unsteady, and that the mother now states that there is no family history of fits, and that the girl "uses both her hands; she used to use the left hand more," but that the mother does not remember when she first noticed the use of the left hand. Probably if the child had been born left-handed the mother would have remembered the fact. Everything seems to point to the fact that the girl was, until the accident, right-handed. The real evidence of this is the fact that she could hardly write legibly, when she was examined by the doctor at 9½ years of age; such a fact would undoubtedly have been noticed at her former schools. When tested in December, 1919, the following results were recorded: Aged 13 years 8 months; rubs left, throws left, cuts left and right, picks up left, winds left; dynam. left = 38, right = 44; writes right but wants to write left.

She wrote her name mirror fashion with her left hand. Unfortunately she was of course never tested in these things before the accident, but it is extremely unlikely that she was then either left-handed, or wrote mirror fashion (as only 4 or 5 in 1,000 do so). She now writes very well with her right hand, and it will be interesting to discover whether she changes in any of the tests as she grows older. The average grip for normal girls is as follows: for 14 years of age, right 24·79; left 22·92.[1] Her right and left hand grips are therefore far above normal.

[1] Smedley's investigations [28].

S. S. writes with the right hand, but "used to wish to write with the left, owing to paralysis of right hand; the paralysis has much improved." When tested, he rubbed, threw, and picked up with the left; he cut easily with his left, but not very well with his right hand. This, therefore, is the case of a boy who was slightly paralysed, but not so badly as to prevent him writing with his right hand.

A. H. G., a boy, aged 6 years. Had a very serious accident when 9 months old from burning, and was not out of danger for over three months; was noticed by mother and nurse to be very left-handed before and after the accident. An intelligent child: at $4\frac{1}{2}$ years of age began to stammer badly with twitching of eyes; both continued until $5\frac{1}{4}$, when the stammer gradually got less and finally disappeared. At 6 years of age he gave hardly any evidences of left-handedness, except that of holding a glass in his left hand while drinking: he always held a bat in his left hand when knocking about a ball. At 7 years old he appeared to be thoroughly right-handed except in one or two small things. Instruments were used at his birth. It is suggested that some cause affected temporarily the hand centre, &c., and that the passing away of this allowed him to revert to his natural handedness.[1]

One or two further references in connection with this question of dominant hemisphere may be of interest.

James Kerr, 1920 [16], writes: "The right hand has generally a greater capacity for fine adjustments and co-ordinations than the left, which last is then accessory. There is at present no worthy explanation of why the right hand should be so specially used, except that it is driven directly from the left side of the brain, and that in the majority of all races that side gives evidence of a higher state of functional evolution than the other." "The interpretation of sights and sounds into ideas and the intellectual functions which control the voluntary machinery of the organism are commonly bound up with functioning of a highly developed left cerebral hemisphere, so that in normal individuals the left hand, although its motor centres are on the right side, is still driven indirectly through these right-sided cortical centres by the higher control of the left side." And again: "The specialized aptitude or capacity associated with psychical phenomena of speech and reasoning which is so marked in the left brain is best described as dextrality. In a small proportion of individuals the reverse happens: instead of the usual high functional development of the psychic qualities in the left brain it is the right side which shows this property; a consequence appears that the nearest motor centres, those of the

[1] "In these cases of congenital hemiplegia and decided left-handedness (which I believe is not infrequently the result of injury to the left cortex). . . ."—G. Vivian Poore [1].

left hand, become the most specially used and the right remains accessory."

Josefa Ioteyko (1917) [10] asserts that it has been proved that the asymmetry extends not only to the centre of projection, but to centres of language, recognition of objects and signs, purposive activity (the loss of which results in ataxia), and that the localization of these centres is in the left hemisphere for right-handed people.

Further it appears to be true that there is an increase in the percentage of left-handed, beginning with normal children in elementary schools (7 per cent.), increasing in the case of feeble-minded to 18 per cent. and rising to 16 per cent. to 30 per cent. in the case of idiots and epileptics.[1]

In connection with the above statements some very curious facts were noted during the present inquiry, facts which at the time appeared to be very contradictory. Very frequently the teachers in ordinary schools asserted that the children found to be left-handed were the most intelligent and the best at school work; in fact some were stated to be the best in the school. Equally frequently the teachers in the mental defective schools described the left-handed to be the dullest, and of the lowest grade. Ballard, who has special knowledge of left-handed children, states in 1916 that left-handed children compare favourably with right-handed in intelligence and school attainments, but he adds that left to right "transfers" (i.e., left-handed children made to write with the right hand) are inferior, and have a tendency to stammer.

One very curious remark was made by the head teacher of one of these special schools, and a special note was made of it at the time, as it appeared to be opposed to what was then thought to be likely, namely, that the change of left-handedness to right-handedness was accompanied by harmful results. The head teacher stated that she had frequently noticed a great improvement in a child's intelligence and school work associated with a natural change of left to right-handedness. There is a very high percentage of left-handed in this school, and no efforts are made to bring about a change. A similar change has been noticed in other schools, an example of which is given below[2]:—

E. Y. (aged 8 years 2 months) throws left, cuts left and right, picks up left, winds left. Copied her name mirror fashion with

[1] Mayet in 1902.

[2] Ireland, W. W. (medical superintendent of Scotland National Institution for Imbecile Children, Larbert), notices a similar change of "handedness" among imbeciles [12].

left, as cannot write it spontaneously. Doctor reports: "Talk four years; walk three years; left-handed at age 7 to 8 years." Head teacher reports: "She is changing from left to right hand in writing without any suggestion from the teacher, and she has begun to improve generally."

There seems therefore to be good reason to believe that there are certainly two distinct types of left-handedness:—

(1) Those who are naturally left-handed, i.e., those who have a dominant right hemisphere.

(2) Those who are naturally right-handed, but who have been driven to the use of left hand in some of their activities owing to some defect of the nervous or muscular systems.

In the case of certain children in the mental defective schools, probably something more than the hand centre or its nerve fibres, &c., has been involved. It also seems probable that the percentage of naturally left-handed in mental defective schools may be no higher than that in ordinary schools, the increased percentage being due to some cause that has brought about the change, before birth, at birth, or at some subsequent date.

These conclusions, as will be remembered, had their origin in the discovery that mirror writing was very prevalent among left-handed children in mental defective schools. A further discussion on mirror writing may therefore be of value. The following are some of the conclusions of other investigators on the subject:—

"In 200 apparently healthy people, only the neuropathic wrote mirror fashion; in 200 sick subjects only those suffering from nervous disorders wrote mirror fashion" [15]. Soltman found (number tested 77) 35 per cent. were mirror writers among deaf-mutes.

Kerr [16] states: "Mirror writing also occurs pathologically with the right hand, being common among hemiplegics and others with brain disease, although even here unusual with better class or well educated individuals. It is to be observed frequently in the developing child. Children becoming deaf soon after birth are said to mirror generally with the left hand, but if the deafness supervenes later the mirror writing does not occur."

McHattie quotes that mirror writing is frequently observed in nervous diseases, e.g., hemiplegia, hysteria, and locomotor ataxy. Among his own observations he gives the case of a girl whose right arm had been partially disabled by infantile paralysis, and who could not in consequence write with her right hand [20].

J. K. Fuller [7] (1916) gives four cases of subjects under hypnotism, three of whom wrote mirror fashion with left hand; one of these is definitely stated to have no inclination to left-handedness.

C. H. Judd also states that "Certain persons when hypnotized produce mirror writing with the right hand" [21].

F. T. Allen (1896) [2] says that mirror writing was discovered when nervous disease rendered normal writing difficult or impossible, and he concludes that it is often a symptom of nervous disease.

F. B. Sherlock [24] writes: "One finds rather widely prevalent the notion that a characteristic of idiocy is that whereas the normal person writes from left to right and with the right hand, the idiot writes from right to left with the left hand . . . the script is meaningless until it is held before a mirror.

"Good examples of this kind of writing are rare, at any rate in this country (England); for idiots do not write at all, or they produce a scrawl, which demands the use of a good deal of ingenuity, in addition to a mirror, if anything is to be made of it.

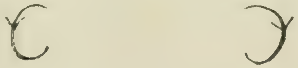
"Ambidexterity, as we shall see, is commoner in idiots than in normal persons, and the particular accomplishment which we are considering seems to be a special case of it."

Although the following quotation (Kerr [16]) does not specify mirror writing, it seems to indicate that such peculiarities may be expected in such a class.

"Whilst in the last class (i.e., defective functioning of both hands) are individuals whose dextrality has not been properly developed, and who are in this sense mentally defective, and on further examination may be expected to present not merely gaucherie, but speech defects, and the various degenerative stigmata in relatively large proportions."

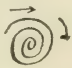
Various explanations have been given as to the cause of mirror writing, but none of them are altogether satisfactory, and they do not account for many of the known facts.

There is no doubt a tendency for the left hand to move in a way similar to movements made by the right hand, e.g., in the simple case of drawing two circles at the same time—one with the right, the other with the left hand, as is illustrated below:—

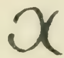


Left. Right

Some experiments were made in the ordinary elementary schools on the effect on the left hand of right-hand practice. Young children 2 to 3 years of age when they begin to use pencil or chalk usually draw a series of circles, thus :—



The direction with the right hand is as indicated by the arrows, i.e. clockwise. If the pencil is held in the left hand the direction is exactly the opposite, i.e., anti-clockwise. The first fact is well known in infant schools, as teachers have some difficulty in persuading children to write an "a" in the usual way, since nearly all begin by writing it as follows :—

 then adding  which gives 

instead of  and  as is usual among older children.

The following results were obtained by asking each child to copy a circle, drawn on a piece of paper or millboard placed before them, first with the right and then with the left hand. Care was taken that the children should not see what had been done by the others, and should not know what they were to draw until they were asked to do it.

Circle drawing with the left and right hand. Note, "N" indicates the natural way, i.e., clockwise with the right, anti-clockwise with left. "A" indicates the artificial way, i.e., anti-clockwise with the right, clockwise with left.

The results obtained with the right hand will be given first.

CIRCLE DRAWING WITH THE RIGHT HAND.

		Percentage of drawing in artificial way
Infant school, average age 4 years 4 months ..	33 children ..	27·2 per cent.
,, ,, ,, 4 to 5 years .. about	40 ,, ..	15·0 ,,

These had not been taught to write.

Infant school, average age 6 years 5 months ..	37 children ..	81·0 per cent.
,, ,, ,, 7 ,, 5 ,, ..	55 ,, ..	92·7 ,,
Boys' school, Class I, average age 12 years 5 months	40 ,, ..	100·0 ,,

That is to say, these children showed a gradual change from the natural to the artificial way of drawing a circle on paper or millboard.

This change was undoubtedly brought about by the continued practice, in writing, of such letters as a, o, d, g, &c. The drawing of circles on the blackboard gives however different results, no doubt in part because it is an arm movement. In the above experiments, and in those that follow, the results given in the case of the babies (i.e., under 5 years of age) are very variable, and not very trustworthy. It would have been much better to have watched these children, and to have noted from time to time how in their ordinary drawing they formed a circle, as it often seemed a mere chance which way they drew the circle; sometimes due to the position of the hand, at others to that of the millboard. The same kind of thing was even more noticeable with their left-hand drawing.

Drawing Circles with Left Hand.

		Percentage who drew circle in artificial way
Infant school, average age 4 years 4 months ..	33 children ..	36·3 per cent. (not very reliable)
,, ,, ,, 6 ,, 5 ,, ..	37 ,, ..	21·6 per cent.
,, ,, ,, 7 ,, 5 ,, ..	55 ,, ..	21·8 ,,
Boys' ,, ,, 12 ,, 5 ,, ..	40 ,, ..	40·0 ,,

More often than not those who drew in the artificial way with the right still drew in the natural way with the left.

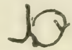
From these insufficient data however it would appear that in some cases the artifical way of drawing with the right had affected the left-hand drawing, as the children (classes ages 6 years 5 months and 7 years 5 months) who drew in the artificial way with the left were those who had also drawn in the artificial way with the right. In these two classes (92 children) only one—a boy—drew in the artificial way with the left, after having drawn the circle in the natural way with his right hand.

The elder boys show a greater change, as the artificial drawing with the left hand has increased to 40 per cent. Whether such a result would be confirmed by further experiments remains to be seen.

There is evidently a tendency, especially when both hands are used at once, for one hand to follow the other mirror fashion. This fact, however, in no way explains mirror writing of children when they begin to write, many examples of which are well known. As a matter of fact an "a" written by the left hand in the ordinary writing is in accordance with the natural tendency of that hand.

Thus  and completed 

To write this letter mirror fashion with the left hand would involve the forming of the circle in the artificial way with that hand, thus :—

 and completing thus : 

As a matter of fact, a fair number of mirror writers did actually write the letters "a" and "o" mirror fashion with the left, but the circle part was drawn in the natural way, i.e.,  .

A widely accepted explanation of mirror writing is given below, but it certainly does not explain all the facts ; nor does it explain why so many more so-called left-handed children write this way with their left hand, unless it is suggested that left-handed children are more motor than visual inclined.

"If a man is of the so-called 'visual' type, i.e., if he depends mainly on his v series in his writing, the *look* of the letters, &c., and by comparing it with the resulting writing, conforming his movement series to it, then any movements which violate the figure presented by visual memory are unintelligible. Such a man must reproduce, with his left hand, the visual images as produced by the right. That is, he must write from left to right, with both hands, which involves symmetrical movements. This represents the power of the v series to bring the movements of both hands in conformity to it. If, on the contrary, his m (motor) series has grown independent by practice, and he remembers written words not by the way they look mainly, but by the way it feels to write them—if he is the so-called motor type in his hand writing—then his left-hand writing must produce the series of muscular sensations as his right hand has established. This represents the power of movements established by one hand to carry the other hand also with it in a symmetrical way. His left-hand position must duplicate at each moment his right-hand position, when he comes to try the experiment of writing in the air with both hands. This gives symmetrical movements with the two hands, which means mirror writing with the left hand" [1] [2].

This symmetrical movement of the two hands has been held by Fechner and others to be a strong proof that the discharge of energy into one side of the body tends to stimulate the corresponding members of the other side to similar movements. Lochte [18] and Weber [27]

[1] J. M. Baldwin : "Mental Development in the Child and in the Race" (1895). A somewhat similar explanation was given by Goldscheider in 1892 [9].

suggest that writing is primarily a motor act controlled by the optical image, which is a later development. Consequently if the mind and the eye are centred on the right hand, we should expect the left hand to follow out the movements of the right hand, and therefore write mirror fashion.

E. J. Allen [2] remarks that the sensations accompanying all kinds of writing, whether right-handed or left-handed, are so similar as to suggest that they all start from the same region of the brain, but that there are a series of commutators at a lower level whereby impulses are turned into different channels. He further suggests that this supports the view that the true graphic centre coincides with either motor centre, but is superior to all.

Josefa Ioteyko (1919) [10] concludes from experiments made in her laboratory by Mlle. V. Kipiani, " . . . in the first place, that the movements acquired by the left hemisphere (which directs the movements of the right hand) are transmitted to the right hemisphere, and, secondly, that this transmission is not purely and simply an increase of excitability, but that it comprises all the complicated movements necessitated by the act of writing—in a word, that the characteristics of hand-writing certainly have their origin in some central cerebral cause."

Before considering further the hypothesis as suggested by Baldwin (p. 353) some examples will be given of mirror writing associated with various forms of paralysis, and with other physical defects.

M. S., aged 13 years 10 months, girl: "Mother in an asylum; in good health until 6 years of age, then a fit with paralysis, right hemiplegia. Reading nil; easy numbers, mentally only; can write fairly well with her left hand: no memory; speech slow and drawling (September 13, 1916); a few letters and words (September 6, 8, June 17). Sleepy, dull and exceedingly slow even for a mental defective; cannot spell "cat" (June 17). "Talks well about her own affairs." The doctor in his last report dated December 19, 1919, states that she is "word blind."

In all the tests that could be tried, she was of course thoroughly left-handed; she could cut easily with her left hand; her strength of grip was 30 kg.

At the first visit all the children in her class were asked to write their names with their left hand and then with their right. Her paper when given up had her name well written at the left side; but there was near the right margin a curious scribble, and emerging from it her name written mirror fashion; the writing was rather shaky. It was originally believed to have been written with her right hand. On a subsequent visit she was watched while she wrote; she immediately wrote her name with her left hand, beginning

in the usual place; the pencil was then placed in her right hand, she had difficulty in holding and using it, but tried her utmost. After scribbling with an arm movement, she took the pencil in her left hand and wrote her name mirror fashion, rapidly and in excellent fashion. She was not known at the school to have written mirror fashion before.

L. C., girl, born April 20, 1907: infantile paralysis, very small for her age. Doctor reports paralysis of right leg and arm; writes mirror fashion with left hand; had given up mirror writing in April, 1916 (i.e., 9 years old); in 1917 (i.e., 10 years old) writes mirror fashion sometimes. January 19, 1920, when asked to try to write her name on the blackboard with her right hand, printed it very well, but all mirror fashion.

Mirror Writers in Physical Defective Schools.

M. A., girl, paralysed both legs. Mentally defective; tested: rubs and throws left; cuts left and right; wrote mirror fashion with her left hand.

J. S., aged 13 years 8 months, boy. Infantile paralysis right lower limb. Nervous. Cuts left and right; writes right. Doctor reports: "Leg flail-like though not very wasted." This boy tried twice not to write mirror fashion with left, but finally wrote both names completely mirror fashion.

A. S., boy, aged 11 years 3 months. Rubs and throws left, cuts left only; writes right, but sometimes left; is thought to be verging on feeble-mindedness. Doctor reports: "Right hemiplegia; slight weakness of right arm and leg, not wasting. Used to fall about a good deal."

J. S., boy, aged 14 years 1 month. Apparently right-handed, mirror writing very shaky indeed. Doctor reports, at 7 years 3 months of age: "Spastic paraplegia (partial); spinal paralysis; right ptosis; very intelligent; at 10 years 2 months, paralysis of eye and legs, getting worse; scissors legs." At present time school staff reports: "Not very intelligent."

E. G., boy, left-hand writer. Writes mirror fashion sometimes also 21 for 12, i.e., beginning with 1 and then writing 2 on the left of it; hydrocephalous.

C. R., boy, aged 8 years 3 months. Writes mirror fashion with left hand when tired. Doctor reports at 5 years 11 months: "Spina bifida, lower cervical region; night terrors; at 8 years 2 months, very backward: spina bifida apparently closed."

J. P., boy, aged 15 years 2 months. Throws left but writes right only: dynamometer left = 34, right 26. Doctor reports at age of 6 years 5 months: "(Brother dead, suffered from fits). Spinal caries (lumbar region), can pick up things. At 11 years 5 months pains in right leg. At 7 years 11 months no active disease; much lordosis, apparently habit."

F. B., boy, aged 8 years 4 months; very large head. Doctor reports: "Infantile paralysis at 2 years 11 months: paralysis of both legs; right arm and leg not bad; operation for talipes. December, 1916, marked wasting of leg and thigh muscles. October, 1919, operation for tubercular disease in third metatarsal bone, sinus still discharging. Bright child."

F. S., boy, aged 10 years 11 months. Right-handed in all tests. Dynamometer, left = 33, right = 32. Doctor reports: "Spinal caries, lower dorsal, at $4\frac{1}{2}$ years."

These nine, together with one left-handed girl suffering from mitral stenosis, who had been in a mental defective school for three years, were the only mirror writers among 706 children tested. It will be noticed that three of these ten were merging on mental deficiency, leaving seven among the physical defective children, i.e., 1·1 per cent. It is remarkable that in six cases there is some spinal lesion, or paralysis of the lower limbs; the seventh is that of a hydrocephalous child.

As reference will be made later to mirror writing by children, when they first began to write, the following cases are given:—

EXAMPLES OF CHILDREN WHOSE FIRST ATTEMPTS AT WRITING WERE WITH THE LEFT HAND AND WERE REPORTED TO HAVE BEEN MIRROR FASHION.

	Age	Sex	Rub	Throw	Cut	Writes	Doctor's report
	Yrs. mths.						
W. S...	..	Boy	R.	L.	O.	L.	
H. S. ..	10 6	Boy	L.	L.	L.	L. and R.	"Bossed forehead; stunted; speech indistinct"
H. W.	8 8	Boy	L.	L.	L.R.	L.	"Speech inarticulate; decided epicanthus; mental age IV. (Binet)"
C. B. ..	10 8	Girl	L.	L.	L.	L.	"Defect of articulation; low bulging forehead"

Similar cases reported by other investigators.

A. L. Beeley [6], 1918, reports and gives details of three boys and two girls, all of whom wrote mirror fashion when they first attempted writing. They were all extremely left-handed and had, with one exception, left-handed near relatives. One, a boy, had not overcome the habit at the age of 7 years 11 months, as "he still frequently lapses into mirror writing."

If there is any truth in the suggestion that those who write mirror fashion with their left hand in mental defective schools are really right-handed (with a dominant left hemisphere), but have some defect in this hemisphere or in the nerve fibres connecting it with the opposite side of the body, a defect which has temporarily or permanently affected the children, then the few who, although they wrote mirror fashion with their left hand, yet in nearly every test reacted right-handedly, would probably display some left-handed activities in some of

their habits—activities, however, that had been insufficient to classify them among the left-handed.

The actual number of those children was five boys and eight girls. It may therefore be asked whether there is any evidence that these children had at any time used the left hand as the dominant hand in any of their activities, that is, whether they had passed through a left-handed stage and afterwards reverted to the right hand.

The following cases are collected from notes made at the schools; in some instances, however, the data are rather meagre, as at first little was attempted beyond the determination of the percentage of left-handed children in these schools.

Mirror writers who appeared to be right-handed :—

(1) L. C., girl: very good mirror writer: rubbed left or right, kicked persistently with the left foot, but in everything else reacted right-handedly.

(2) B. C., girl: a very good mirror writer; reacted right-handedly to every test, except (1) dynamometer, left = 36, right = 34; (2) a special note was made of her holding her left arm stiffly when catching.

(3) R. R., girl: (1) rubbed with left but afterwards with right; (2) held her left arm down when catching; (3) dynamometer, left = 38, right = 32.

(4) W. S., boy: mirror writing poor; reacted right-handedly in everything except the dynamometer, left = 39, right = 25.

(5) G. S., boy: hydrocephalous with very little power in his legs; reacted right-handedly in everything except "winding," and dynamometer, left = 15, right = 12.

(6) E. T., boy: reacted right-handedly in everything, but held his left arm down characteristically while catching.

(7) H. L., girl: right-handed in everything except grip test; dynamometer, left = 30, right = 19.

(8 and 9) G. C., boy; F. C., girl: no left-handed indications, but wrote mirror fashion with left hand very well.

(10) M. J., girl: no indications, but she copied from a book a whole page correctly and rapidly with her left hand.

Of course it may be argued that every child may do something left-handedly, provided a sufficient number of tests are tried. The results of these tests are given, but too much weight must not therefore be attached to any conclusions that may be drawn from them. There were certainly a few cases where the children who wrote mirror fashion with the left hand gave no indications of left-handedness. It seems not impossible that some at least of the above children may have acquired left-handed habits at one time of their life, but that only one or two of these habits may have persisted. Why some habits may

persist, and others be lost, it is difficult to say, but one example is very illuminating :—

L. F., a girl: mirror writer, reacted right-handedly, but only cuts easily with her left hand with the special scissors. She always sews and knits left-handedly. It seems evident that she must have been left-handed, when she learnt to knit, as nearly all teachers try to make the girls right-handed in knitting and sewing. Having once learnt to knit left-handedly, it can be easily understood why she continued to do so, although she may have changed into right-handedness in everything else. She suffers from epileptic fits.

Before drawing any further conclusions from the facts as given, it is very necessary to examine more carefully the theory as stated by Baldwin (p. 353) to see whether the facts in reference to mirror writing may not be sufficiently explained by such a comparatively simple hypothesis.

The hypothesis is in short that in the case of those who are of the so-called motor type in hand-writing the left hand will reproduce a series of muscular sensations as has been established by the right hand, that is, that they will remember written words not by the way they look mainly, but by the way it feels to write them.

Undoubtedly in drawing a line or a circle the tendency while using the right hand is to draw the line from left to right, and the circle clockwise. A similar feeling will be experienced by the left hand in drawing a line from right to left, and a circle anti-clockwise. This tendency would undoubtedly explain many cases of mirror writing, but it certainly does not seem to explain :—

(1) Why so many mirror writers in mental defective schools are very left-handed in many of their activities.

(2) Why some children write mirror fashion when they first begin to write, as no muscular sensations can have been established in such cases. Of course the same argument holds in the examples of mirror-writing with the right hand.

(3) Why so many mirror writers, although they now write with the right hand, at one time wrote with the left. It would seem probable that any practice in writing with the left hand would have tended to correct an inclination to write mirror fashion with same hand at a later period, and that these children would therefore be less liable to such kind of writing than the right-handed child, who writes with the left hand for the first time.

(4) Why a girl with the right upper limb paralysed at 6 years of age, who had written with her left hand in the ordinary way for nearly

eight years, should suddenly write mirror fashion with the same hand, after making an effort with her right hand.

Also other cases of mirror writing by left-hand writers who had suffered from right hemiplegia from an early age.

(5) Why "certain persons when hypnotized produce mirror writing with the right hand." Should this statement be true, only one explanation seems possible, if it is to be in accordance with the above hypothesis, namely, that these "subjects" were really left-hand writers.

There also seems no reason to believe that left-handed children (even among the feeble-minded) should be more frequently of the "motor" than the so-called "visual" type. There is however this to be said, that a child with a fairly skilful left hand may be able to write with this hand without paying so much attention to what is actually being written, and for this reason might be inclined to trust to a feeling of the movements of the hand rather than to the actual forms of the letters and words written. On the other hand it has been pointed out that some children have actually attempted to correct their movements, thus clearly indicating that they had noticed that there was something wrong. These attempts, however, were not always successful. One or two examples of such attempts are given below.

(1) A boy wrote the first letter of his name mirror fashion; he then started again and wrote the first two letters of his name in the same way. Even after these two unsuccessful attempts he finished by writing both his Christian and surname completely mirror fashion.

(2) Two or three children at least wrote their names or parts of their names mirror fashion, and then turned over the paper and had another try, but with no better success. It would seem that these children had noticed what they had done, but could not make their left hand do what they wished or expected.

We might, however, supplement the hypothesis under discussion by supposing that there is no real necessity for assuming an established series of muscular sensations, that are then reproduced by the left hand in a "reflected" manner (i.e., mirror fashion). For we can go this far and suggest that quite apart from the establishment of such a muscular feeling there is a "natural" direction of writing with the left hand and that it is from right to left, and that this natural direction may quite well involve a tendency to form letters and even words in a manner the reverse of the natural way with the right; that is to say, that even before letters and words have been copied by the right hand there will be an inclination for the left hand to write mirror fashion

unless corrected by visual sensations. Suppose therefore mirror writing is the more natural method of writing with the left hand, but the tendency is corrected by the look of the letters and words, then the first writing, before the correlation of the hand and eye has been perfected, may well be mirror fashion, as has been found in some cases. But such an explanation is negatived by mirror writing with the right hand by beginners.

Again, the original theory as stated by Baldwin might explain in a simple manner why the feeble-minded were more prone than normal children to write mirror fashion with the left hand, after they had learned to write with the right. It would seem quite justifiable to suppose that mirror writing by the hand not habitually used in writing is the automatic reproduction by that hand of the ordinary writing, and that it is due to some mental defect, probably a defect of the higher centres (involving voluntary action), the disarrangement of which has let loose the more automatic "activity of the lower centres, or the nervous arrangements which have escaped injury," and has thus allowed the unpractised hand to follow the more automatic series of movements.[1] In the case of hypnotized "subjects" such an explanation would equally apply, provided the mirror writing was done by the hand not habitually used in writing, but it would of course be negatived by mirror writing with the right hand by right-handed "subjects." There is further evidence in support of this explanation in the case of children who lapse into mirror writing when inattentive. Beeley [6] gives the following examples :—

(1) Boy aged 8 years 3 months; bright, lapses into mirror writing when inattentive, knows the difference, very good memory, left-handed writer, left-handed family.

(2) Boy, aged 7 years 11 months: not yet overcome tendency to mirror writing; when his attention shifts from the "executive factor" to the "thought element" lapses to mirror writing. Stated to have written mirror fashion from the beginning, at 4½ years of age, and not to have recognized that it was wrong.

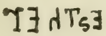
Other experiments were made with letters cut out of stiff paper and placed on the table the reverse side upwards, to see whether mirror

[1] If this is a true explanation one would expect mirror writing to be frequently found in the numerous cases of lesions which have affected the higher centres, and have been associated with aphasia and right hemiplegia. But this appears to be very seldom the case among adults; in fact Moutier gives only two examples of mirror writing among all the cases in his book [22] [Nos. 361 and 172].

writers would arrange them from right to left or attempt to arrange them in the correct direction, i.e., from left to right.

L. J., girl, aged 11 years 10 months: left-handed in all the tests, but writes with the right hand, and wrote mirror fashion with the left at a previous visit. When given the mirrored letters, and asked to form with them the words "cat" and "dog," using her left hand, she arranged them without any hesitation mirror fashion, i.e., from right to left. The same letters were then rapidly and without her knowledge turned the right way up; she now arranged them with the same hand in the correct position. Exactly the same result was obtained with the right hand. A 4 and 5 were then given her, and she was asked to make 45 with them, using her left hand. She arranged them thus:  , but seemed dissatisfied with the result. After looking at them for a short time, she took the 5 and placed it on the right side of the 4, thus:  .

The following case seems to indicate that the mirrored letters are sometimes at all events a reproduction of an optical memory image, and not a mechanical reversal of what has just been seen.

E. Y., girl, aged 8 years 1 month: reported to be changing spontaneously from left to right in writing. Left handed in all the tests. As she could not write her name spontaneously it was printed, and she was asked to copy it with her left hand. The name was printed thus: "Esther." She copied it as follows: —that is, she had changed the small "t" and "e" into capitals "T" and "E."

The curious case of children printing with their right, and writing mirror fashion in a "running" hand with the left, and also those who write mirror fashion slowly and obviously looking at each letter as it is being written, seems to indicate that it is a reproduction of an optical image.

Kerr states: "Similarly infants, tested with copying English and Russian words, only made mistakes with letters common to both alphabets—that is, those with whose forms they had some slight acquaintance—but copied correctly the complicated letters peculiar to Russian, whose form they had never seen before" [16].

From such facts it would seem that something had affected the nerve fibres connecting the hypothetical visual memory of words centre with other parts of the brain; this centre, if there is such a one, is generally believed to be situated in the dominant hemisphere. If this

should be the case, the association of left-handedness and mirror writing might be explained by some lesion that has affected the dominant side of the brain, or by a lack of development of this side—resulting in some form of mental deficiency, in a change of right to left-handedness, and in mirror writing.

Chapter VI.—Defective Speech and Left-handedness.

Ballard and others [5] [14] have shown that stammering is more prevalent among left-handed children, who write with the right hand, than among "pure" left or right-handed children. Also Lewis [17] has reported that epileptic children—originally right-handed—first hesitated in speech, and then developed a decided stammer, after being trained to write with the left hand in the ordinary way, i.e., in the direction left to right. These same children had previously shown no indication of hesitating in speech, although they had been practising writing with the left hand for two or three months, but the writing had been mirror fashion and from right to left in direction. That is, a change in direction of the writing with the left hand did not seem to affect the speech, but when the direction was unchanged then the speech appeared to be affected. This is also what appears to be the case with some left-hand writers who have changed to the right hand, but of course no experiments have been made with left-hand writers to see whether mirror writing with the right hand would have the same or a different result.

During the present investigation many instances of the same kind were noticed among normal children, but it appeared that among mental defectives there were just as many stammerers among left-handed writers as among left-handed children who wrote with the right hand. In addition to this, defective articulation and other speech defects seemed to be prevalent among mirror writers. No special note was at first made of such defects, as it did not seem probable that the association of mirror writing with defects of the speech was anything more than a coincidence. Later, however, much greater importance was attached to these facts, especially when it seemed likely that many of the children with left-handed activities might be right-handed children (i.e., with a dominant left hemisphere), children who had been driven to the use of the left hand by some defect of the left hemisphere, or the nerve fibres connecting this hemisphere with the right side of the body. As (p. 343) paralysis of the right side

(right hemiplegia) is frequently associated with aphasia, and it is therefore possible that with these mental defectives the same defect may have affected not only the hand but also the speech of these children. The following cases are of interest in connection with this subject.

(1) M. G., girl, born March, 1911. Doctor reports : "Meningitis at 2 years ; speech defect the most prominent feature ; left-handed. February, 1918 : ditto, and poor at manual work, October, 1919." She was tested April, 1920 : rubs right, throws right, cuts left and right, writes right. The head teacher reports : "She uses her left hand at times in some things."

(2) E. H., girl, born March, 1910. Doctor reports : "Mother says : 'lost her speech for eleven days, and was like an idiot for a week.'"

Tested April, 1920 : Throws left, cuts left, picks up left, writes right and left ; knits left ; dyn., left 13, right 15.

(3) J. P., boy, born February, 1907. Doctor reports : "Premature birth 7 months ; drags right leg ; ? hemiplegia ; talipes ; right hand less powerful than left ; slight defect of speech ; left handed January, 1916 ; stammers January, 1918 ; slightly deaf, otorrhœa (right ear) ; slight paresis of legs."

Tested April, 1920 : He still stammers. Throws left, cuts left and right, picks up left, winds left, writes left. This is interesting, as it seems to be a case of slight right hemiplegia with a slight speech defect, which developed into a stammer.

Speech Defects among Mental Defective Children.

	Left-handed		Right-handed	Total
	Write with Left hand	Write with Right hand		
Stammer or stutter	19	20	44	83
Defective articulation..	14	1	18	33
Defective speech, including some cases of defective articulation	39	27	40	106
Lisping associated with other speech defects	6	1	7	14
Great hesitation in speech	0	2	1	3
	78	51	110	239
Percentage ..	32·6 %	21·3 %	46·0 %	99·9 %
	53·9 %			

The above statistics have been made out in connection with children with very defective speech ; in every case they have been tested in "handedness." Now, as there are about 81·8 per cent. right-handed among mental defectives, and 18·2 per cent. with many left-handed activities, there are, therefore, four and a half times as many right-handed as left-handed, and if very defective speech is not associated with left-handedness, then there should be among these children

with very defective speech four and a half times as many right-handed as left-handed. The results of an investigation on this subject are given above. The numbers are not large, and therefore the result can only be treated as provisional. Cases in which the causes of the defect seem to be clear have been omitted, e.g., split palate, bad adenoids, &c. The number of cases was 239; all the children stammered or had stammered, or were stated to be bad cases of defective articulation or of defective speech.

From the above it will be seen that taking the various speech defects (239), more than half are left-handed, whereas one would expect about 18 per cent. or so to be left-handed unless there was some connection between left-handedness and speech defects. The number of stammerers among the left-handed and among left-handed children who write with the right hand are approximately equal, i.e., nineteen and twenty respectively. These statistics deal exclusively with mental defective children, and it is probable that the same result would not be obtained with normal children. If this is the case, and it seems likely from the results of previous investigators, then it becomes a matter of great importance, as it would seem to indicate that there is something quite distinct in the left-handedness of children under investigation, and that this left-handedness is frequently associated with speech defects.

An interesting case not included in the above is that of a child with defective speech :—

A. W., girl, aged 15 years 8 months: was paralysed (left arm and side) at $2\frac{1}{2}$ years of age. She does everything right-handedly, and cannot even hold a pencil in her left hand. It seemed likely that she was originally left-handed, and that the paralysis had affected her dominant (right) hemisphere, as she is in a mental defective school and her speech is defective. Her mother was therefore questioned, and she stated that "the girl was left-handed until she had the paralytic stroke."

Summary and Conclusions.

Left-handedness.

(1) The percentage of left-handed children (i.e., with marked left-handed activities) is much higher in mental defective schools than in ordinary elementary schools :—

Percentage in ordinary schools	7·3
Percentage in mental defective schools	18·2

Note.—(i) About 7·2 per cent. in mental defective schools write habitually with the left hand; (ii) the percentage of left-handed girls (mental defective schools) is about 25 per cent higher than that of the boys.

(2) Left-handedness in mental defective schools is more frequently associated with defects of the speech than is right-handedness (p. 364).

(3) In the case of twins, when one is left-handed and the other right-handed, the left-handed twin is frequently backward, less developed, highly nervous, or even in a mental defective school, whereas the right-handed twin is normal and in an ordinary elementary school. Among twins (a boy and a girl) there is a very high percentage (nearly one-third) where one is left-handed and the other right-handed.

(4) Among normal children the left-handed are frequently the most efficient and capable; among the mental defectives it is exactly the reverse, left-handedness being often associated with marked deficiency. Spontaneous change of left-handedness to right-handedness in writing among mental defectives is often found to be associated with progress in school work and in intelligence.

Mirror Writing.

(5) The percentage of mirror writers is much higher in mental defective schools than in physical defective schools and in ordinary elementary schools.

	Elementary schools	Physical defective schools	Mental defective schools
Percentage of mirror writers ..	0·48 ..	1·1 ..	8·0

(6) Percentage of mirror writers among left-handed and among right-handed children in mental defective schools:—

	Left-handed	Right-handed
Boys	18 per cent. ..	1·6 per cent.
Girls	40 ,, ..	2·8 ,,
Boys and girls ..	30 ,, ..	2·2 ,,

(7) Percentage of mirror writers among left-handed children who write with the right hand:—

Boys	32 per cent.
Girls	62 ,,

The great majority of mirror writers are children who are reported to have written with the right and also with the left hand, but this is not invariably the case, as many mirror writers, although left-handed in many things, are said to have never written with the left hand; other mirror writers suffering from right-hemiplegia from an early age have never written with the right hand.

(8) Children who are reported to write on some days with the left and on other days with the right hand can frequently be made to write

mirror fashion by merely asking them to begin at the right edge of the paper. Some of these children write in the same way with the right hand; others cannot be induced to write mirror fashion with the right hand. In many cases these children have never been known to write mirror fashion before. Children reported to have written mirror fashion when young can often be induced to write mirror fashion under similar conditions.

(9) Many left-handed children are reported to have written mirror fashion with the left hand when they first began to write and to have persisted for some time. A few right-handed children also wrote mirror-fashion with the right hand when they first began to write.

(10) In no case has a left-handed writer been reported to have written mirror fashion on changing from left to right hand writing. These same children when asked to write with the left hand frequently do so mirror fashion.

(11) In some instances all the letters are written mirror fashion with the exception of one or two letters, and these are persistently written correctly, and with both hands; in the other cases the correct letter (with left hand) is written mirror fashion with the right.

(12) Of the 109 cases of mirror writing, eight (i.e., 7·8 per cent.) wrote mirror fashion spontaneously with the right hand and correctly with the left.

(13) Mirror writing seems in some cases to be due either to (*a*) a motor memory image, or to (*b*) an optical memory image (p. 361).

Theoretical Conclusions.

There are undoubtedly some peculiar and uncommon characteristics (uncommon even among mental defectives) among some of the left-handed children in mental defective schools, especially among those who write with the right hand. The fact that very many of the mirror writers are left-handed is merely a statement of fact and no explanation. There seems no adequate reason for believing that naturally left-handed children (i.e., with a dominant right hemisphere) should be more prone to mirror writing than the naturally right-handed (i.e., with a dominant left hemisphere), nor is there any reason for supposing that naturally left-handed children should be more inclined to suffer from speech defects than the right-handed. In addition to these facts there are undoubtedly a much larger number of left-handed among mentally defective than among normal children, and many of these are of a very low-grade type.

There seems at present to be only one explanation possible, and this explanation would clear up many, if not all, of the observed peculiarities and apparently contradictory facts. It of course does not go to the root of the matter, and much still requires elucidation.

The hypothesis suggested is that something has occurred which has interfered with the proper functioning of the dominant hemisphere—in the majority of cases described it is, of course, the left hemisphere. Such a supposition would explain :—

(1) Why there are so many left-handed in mental defective schools. The left hemisphere being affected has interfered with the proper functioning of the right hand, perhaps in only a slight degree, as many among the older children change spontaneously from left to the right hand in writing. The same cause has probably affected the functioning of many of the higher intellectual centres, supposed to be situated in the same hemisphere, involving such peculiarities as mirror writing and defective speech.

(2) It would also explain why, in the case of left and right-hand twins, the left-handed twin and not the right-handed is found in these special schools.

Whether the hypothesis is true or only partly true, or whether it is untrue, is not of great moment for the present; it has undoubtedly indicated new lines of investigation, and has been fruitful in suggesting fresh experiments and observations.

BIBLIOGRAPHY.

[1] Allbutt's. "System of Medicine," 1899, vol. viii, p. 3.
[2] Allen, E. J. "Mirror Writing," *Brain*, 1896, vol. xix, pp. 385-7.
[3] Baldwin, J. M. "Mental Development in the Child and in the Race," p. 99.
[4] Ballard, P. B. "Left-handedness," *School World*, December, 1916.
[5] *Idem.* "Sinistrality and Speech," 1912.
[6] Beeley, A. L. "Experimental Study of Left-handedness," Supplementary Educational Monographs, University of Chicago, June, 1918, Chicago, 1918.
[7] Fuller, J. K. "Psychology and Physiology of Mirror Writing," University of California, published in *Psychology*, May 8, 1916, vol. ii, No. 3, p. 199.
[8] Galton, F. "Inquiries into Human Faculty," 1911, p. 156.
[9] Goldscheider. *Arch. f. Psychiatr.*, 1892, Bd. xxiv, S. 503.
[10] Ioteyko, Josefa. "The Science of Labour and its Organization," 1919, pp. 96, 126, 143.
[11] Ireland, W. W. *Brain*, 1881-82, vol. iv, p. 361.
[12] *Idem.* *Brain*, 1880-81, vol. iii, p. 207.
[13] Jackson, J. "Ambidexterity," pp. 10, 141, 163.
[14] Jones, Franklin. "A Study of Handedness," University of S. Dakota, series xvii, March, 1918, No. 14.
[15] Jones, H. Macnaughton. "Ambidexterity and Culture," p. 66.

[16] Kerr, James, M.D. "Left-handedness and Mirrored Writing," *School Hygiene*, 1920, vol. xi, p. 39.
[17] Lewis, A. E. "Left-side and Ambidexterity," *Times Educational Supplement*, May 6, 1920.
[18] Lochte. *Arch. f. Psychiat.*, 1896, Bd. xxviii, S. 379.
[19] Lombroso. *North American Review*, 1903, vol. clxxvii, p. 440.
[20] McHattie. "L.C.C. Report of Council," *Public Health*, 1913, vol. iii, p. 120.
[21] Monroe. "Mirror Writing" *Cyclopædia of Education*, 1913, pp. 108, 252.
[22] Moutier, F. "L'Aphasie de Broca," 1908, p. 118.
[23] Schafer. *Berl. klin. Wochenschr.*, 1911, Bd. xlviii, S. 295.
[24] Sherlock, F. B. "The Feeble-minded," 1911, p. 86.
[25] Storck. "Les Stigmates anatomiques et physiologiques de la dégénérescence et les pseudo-stigmates anatomiques et physiologiques de la criminalité," Lyon, 1902, p. 97.
[26] Wallin, J. W. W. Annual Report, Board of Education, St. Louis, 1915-16.
[27] Weber. *Zeitschr. f. Klin. Med.*, 1895, Bd. xxvii, S. 260.
[28] Whipple, A. M. "Manual of Mental and Physical Tests," 1916, part i, pp. 102, 107, 139.

TONUS AND THE REFLEXES.

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[*Delivered before the University of London on January* 17, 1921.]

THE Anglo-Batavian Society put forward the project recently for a series of lectures to be delivered in England by Dutch and in Holland by English medical men. Through the whole-hearted co-operation of the Royal Society of Medicine and the University of London, as well as of the Royal Academy of Amsterdam, this project has become reality. I have been invited to inaugurate this series from the Dutch side. The undoubted temerity of accepting this invitation is only to be explained by my knowledge of the generous judgment and courteous forbearance with which my fellow-workers in the field of science are received in your midst. Yet I fear that I shall have to crave your kindness and indulgence.

The subject of my lecture is one which in Holland is engaging many workers, and on which I have also expended considerable thought and time as well. I mean the question of tonus and reflexes. In my work on this subject I made an extensive use of Einthoven's ingenious invention, the string-galvanometer. I hope to show you a very small part of this work and to submit to your consideration some remarks on the result and conclusions to be drawn from it.

The investigation of the electric response of contracting striped muscle by means of the string-galvanometer has led us to classify the muscle contractions into three different groups. A representative of the first group may be found in the simple muscle twitch, such as is elicited by stimulating a motor nerve of the muscle itself with one condenser discharge or an isolated break-shock from the secondary of an induction coil. The resulting muscle twitch is characterized by its short duration as well as by its being preceded by a double-phase action current. During the contraction itself the muscle is electrically inert. An example of the second group is the voluntary muscle contraction, which is characterized by a current of action in the form of a rather irregular alternating current with a frequency of about 50 periods per second. The electric responses of the first and of the second kind can easily be

recorded in our patients. The last group comprises all kinds of muscular actions in which no electric current of action can be detected.

To these three groups we might possibly add a fourth, comprising contractions caused by special forms of electric stimulation, in which we are able to record a few abnormal forms of electrical response. As these contractions are always artificial and never occur physiologically, and as they have no direct bearing upon our subject, we need not take them into further account.

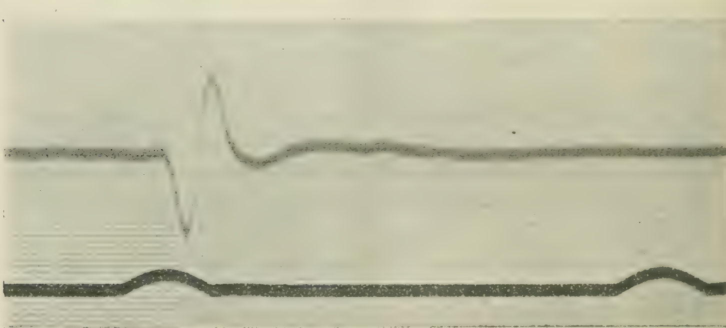

Fig. 1.—Knee reflex. Electromyogram. 400 mm. per second.

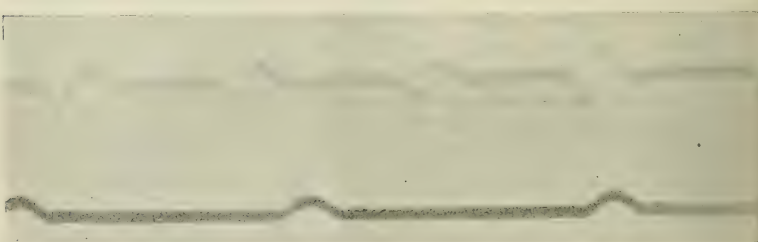

Fig. 2.—Knee clonus in myelitis. 250 mm. per second.

In human physiology and pathology muscle contractions of the first kind, in the form of simple muscle twitches, are to be found, of course, after electric stimulation, but also, as I found in 1907, in all deep reflexes, tendon reflexes as well as periosteal reflexes. In cases of muscle-clonus in lesions of the lateral tracts, I found a regular series of simple twitches, each starting after a complete biphasic action current. Between these

the galvanometer-string practically does not move at all. I have not been able to find a single other instance of the occurrence of simple muscle twitches under physiological or pathological conditions.

The second kind of muscle contraction is met with in every voluntary movement, as was first demonstrated by Piper. The irregular alternating current of some fifty periods per second is the electric phenomenon which

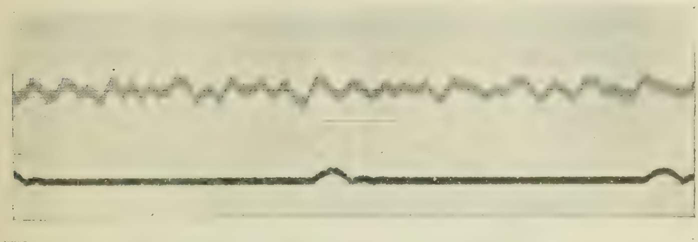

FIG. 3.—Voluntary tetanus. Time marks 0·2 second.

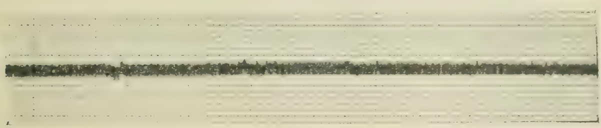

FIG. 4.—Myoclonic-epilepsy. Very short tetanus, nearly resembling simple twitches.

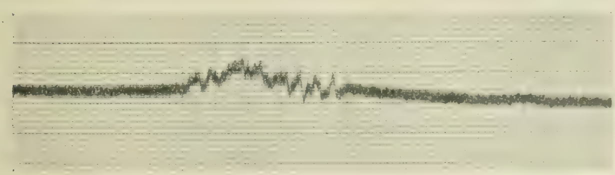

FIG. 5.—Electromyogram in normal plantar reflex. 100 mm. per second.

is typical of the tetanic contraction. If found in any contraction we may be absolutely sure that we have a tetanus before us. In cerebral contraction, such as epileptic convulsions, the string-galvanometer again shows the tetanic character of the muscular shortening. In two cases of myoclonic epilepsy I at first thought that I had at last found single muscle twitches of cerebral origin, but a closer examination of the

galvanometric records proved them to be also short tetani, the shortest I have ever seen. Many other different kinds of muscular action belong to this group, as the examination with the string-galvanometer proves. Such is the case with all cutaneous reflexes, as I had found many years ago. The duration of the tetanus may be very short, as short as 0·1 second, though generally it is much longer, yet we never fail to find the irregular oscillations of the string which indicate that the contraction was a real tetanus.

The third kind of muscle action is perhaps the most interesting. We may observe strong muscle spasms and also notable changes of the muscle tension without any electric action current. All changes in muscle tonus occur in this way and do not seem to cause any change in the electric state of the muscle. If the triceps brachii be connected to the galvanometer-terminals and the arms passively extended or bent, the string does not move. Yet the passive movement of extension is accompanied by a shortening of the muscle, the tonus-contraction. Similarly a tonus-relaxation does not act upon the string. In the case of myelitis with complete paralysis of the lower limbs, we may find enormous muscle spasms which do not show any action current. The same may occur in hemiplegia, lateral sclerosis of the cord, multiple sclerosis or generally in complete degeneration of the pyramidal tracts. I found them also in focal disease of the corpus striatum and regio subthalamica. In all these morbid conditions we have strongly hypertonic muscles. As far as we can judge, the absence of a typical action current seems to be the one and only means of differentiating between a tetanic and a tonic spasm or contraction. We should therefore use the Einthoven instrument in order to discover the nature of any muscle tension about which we are in doubt. Fröhlich and Meyer employed it for this purpose with a patient suffering from tetanus infection and found that the muscle spasms were tonic and not tetanic. I had occasion to examine a cat infected with tetanus by Professor Magnus, and easily obtained a record from the spastic triceps muscle showing a straight line interrupted only by small regular notches produced by the action current of the heart. On the other hand I found with Dusser de Barenne decerebrate rigidity to be a tetanic state. In veratrine poisoning we get a tonic muscle spasm, whilst strychnine poisoning produced real tetani, as was discovered years ago by Lovén and by Burdon Sanderson. Cataleptic muscle spasm is described by Fröhlich and Meyer as being of tonic origin, which does not seem to be in contradiction to an earlier investigation by Gregor and Schilder, whose

curves showed slight irregularities. As regards tetany in children and adults we have as yet no certain data. Two infantile cases which I examined did not yield satisfactory results and left me undecided as to the origin of carpopedal spasms. Yet I incline to the belief that these spasms are of tonic origin.

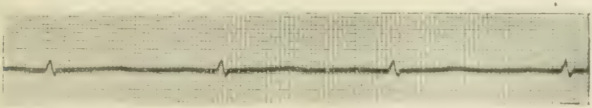

FIG. 6.—Tetanus infection. Electromyogram of triceps. 50 mm. per second.

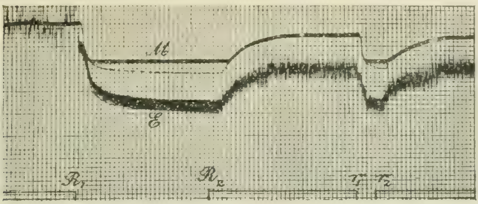

FIG. 7.—Decerebrate rigidity. M = Myogram. E = Electrogram. From R_1—R_2 stimulation of central nerve stump. After Einthoven.

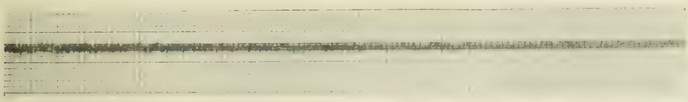

FIG. 8.—Infantile tetany. Action current in flexors of fingers. 100 mm. per second.

The question about the after-contraction in Thomsen's disease seems likewise to have been settled by Gregor and Schilder, who were not able to find the action-current typical of tetanus. I too have failed to find it, and therefore cannot agree with the recently published opinion of Schäffer who, after cessation of the voluntary tetanus, still found slight indications of an irregular oscillatory action-current which gradually tend to subside. I believe that these movements of the string are not caused by the tonically contracting muscles, but by the active contraction of the antagonists, which contract involuntarily to oppose the after-contraction.

An exceedingly curious kind of after-contraction was first described

under the name of "Katatonus-Versuch" by Kohnstamm in the *Neurologisches Zentralblatt* of 1915, but, as far as I know, has not yet penetrated into English medical literature. We can elicit this after-contraction in different muscles, but most easily in the deltoid. To do this, we should first voluntarily cause the muscle to contract isometrically with as much force as possible, and keep this up for perhaps a minute, the muscle being kept stretched all the time by fixing the arm. If then the voluntary impulse be stopped and at the same time the arm be released, we feel that the arm is drawn upwards, without, and even against, our will. Of course, we can easily prevent the arm being lifted, but if we try to remain absolutely passive, refrain from any voluntary impulse and just let the arm do what it seemingly wishes to do, it is lifted. The experiment succeeds equally well with

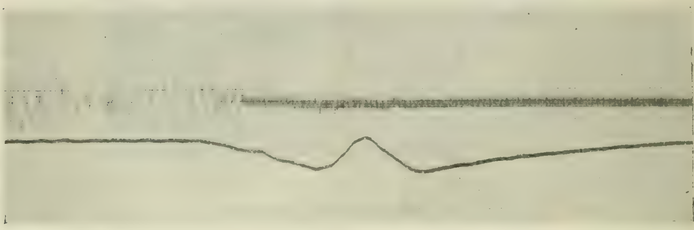

FIG. 9.—Katatonus experiment. Lower curve mechanical myogram. 50 mm. per second.

the hand extensors, whereas it is far less pronounced with most of the other muscles of the body. Csiky has shown that a strong prolonged faradic tetanization, during which the muscles are prevented from shortening, will also cause this after-contraction to appear. The duration of the after-contraction may be from one to ten seconds. This katatonus experiment has been ascribed to various causes. Kohnstamm thinks it may be an involuntary tetanus, whereas Csiky regards it as an idiomuscular contraction. If by this he means the same thing as what I call a tonus-contraction, I can agree with him, for examination with the string-galvanometer shows it to be an undoubted tonus-contraction.

There are many forms of hyperkinesis, regarding which we do not yet know whether they ought to be considered as tetani or tonus spasms. I shall not discuss these here, but shall give you later on the results I obtained in one particular field of research, namely, the reflexes. I have already mentioned that in normal deep reflexes we always get a simple

muscle twitch preceded by a diphasic action current, and that the knee and ankle clonus as well as the hand clonus in cases of sclerosis of the lateral tracts is composed of a regular series of simple twitches, each giving one single diphasic action current. Between each two successive electric impulses the muscle is electrically inert. But how is it with hysterical clonus? Here the galvanometric record shows the presence of a typical tetanus on which is superposed a series of somewhat stronger and probably not entirely regular diphasic action currents. It is generally assumed that the mechanical condition for the appearance of clonus demands an increased reflex-irritability and at the same time the presence of a certain amount of muscle-tension. Only the co-existence of both circumstances renders it possible for the muscle to contract clonically. With degeneration of the lateral columns this increased

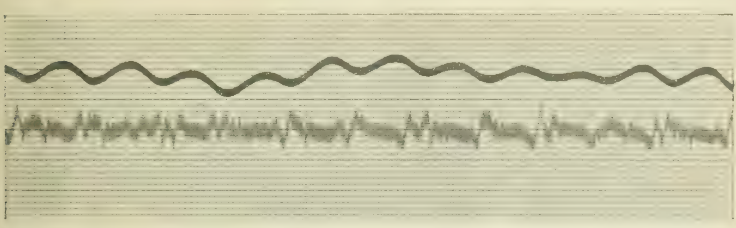

FIG. 10.—Hysterical knee clonus. 100 mm. per second.

muscle-tension is caused by increased tonus, whereas in hysterical clonus this is due to a tetanic muscular contraction. Although in general we shall not have any great difficulty with the differential diagnosis between organic and hysterical clonus, we may with advantage use the string-galvanometer for difficult or doubtful cases. We may probably even go a step further and say that the symptom here mentioned constitutes the only absolute and reliable symptomatical and genetic difference between the two conditions.

Frequently much definite and useful knowledge may be gained by simply recording the mechanical myogram in deep reflexes, especially when they are exaggerated. The normal knee-jerk gives a myogram which may best be compared to one from a gastrocnemius preparation of the frog. But as soon as we have to deal with exaggerated reflexes we hardly ever find this simple form. More often we find clonic contractions either in a short or in a longer series, each of the com-

ponents giving the well-known biphasic action current, in the same way as with the organic knee-clonus. In another group of cases we find that the initial twitch is followed by only one more contraction, which may commence after the principal jerk is entirely finished, but

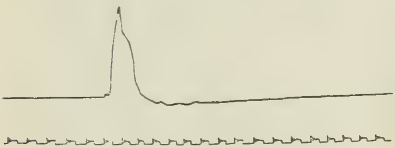

FIG. 11.—Knee reflex, simplest form.

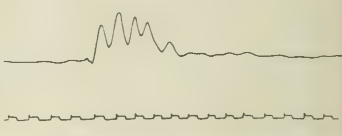

FIG. 12.—Knee reflex, simple clonic response.

FIG. 13.—Knee reflex, clonic after-contractions superposed on tonic secondary contraction.

generally a little earlier. This depends upon the duration of the first twitch, which generally lasts from 0·2 to 0·3 second, the second contraction usually beginning 0·25 to 0·3 second after the moment of percussion. Now, it is curious to notice that these double contractions may occur under widely different circumstances. I shall give a few instances.

We frequently find a double contraction in cases of chorea minor where its occurrence has already been described by Gordon Hey, Eshner, Kleist, generally as a lengthened response. This symptom was considered as a choreic movement following the reflex extension. The graphic record proves that the muscle invariably relaxes at least partially between the two contractions, and furthermore that the last regularly occurs after a definite latent period of 0·21 to 0·25 second. Its duration is not constant. Fahrenkamp has examined the action current and found the second twitch to be a tetanus. I found the same thing, so that we may take it as a fact that the knee-jerk is sometimes immediately followed by a second contraction of tetanic character. In several cases of sclerosis of the pyramidal tract the mechanical myogram of the knee-jerk also showed a double contraction not infrequently of different types.

In one kind the second contraction was an unbroken one of very short duration, although contractions lasting some 10 to 20 seconds may also be met with. In another type it appeared in the form of a series of clonic contractions from anything between two or three and a few hundred. Whereas this last clonic group was found to be electrically defined as an organic clonus, the first group did not give any deflection

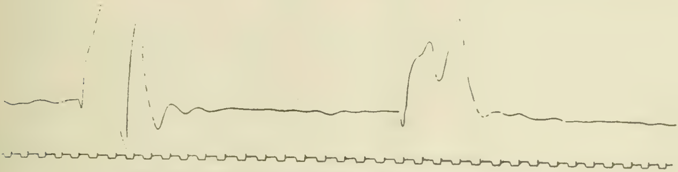

Fig. 14.—Knee-reflex in chorea. Double contraction.

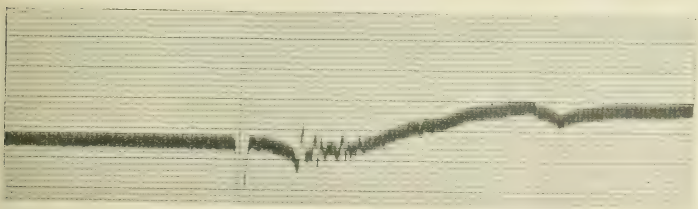

Fig. 15.—Knee reflex in chorea: Gordon Hey type. Action current of quadriceps. 100 mm. per second. Simple twitch followed by tetanus.

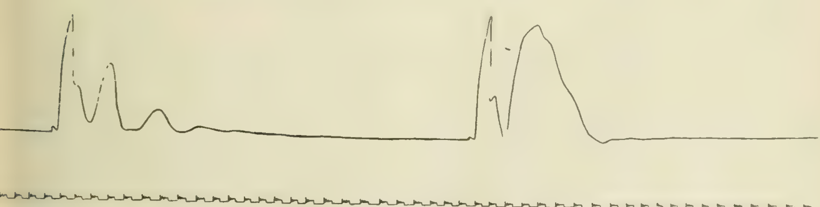

Fig. 16.—Knee-reflex in disseminated sclerosis. Double contraction.

of the string. This implies that the knee-jerk was followed by a longer or shorter tonus-contraction. But a point of interest is that we are able to produce at will in some patients either a tonic or a clonic after-contraction. We do this by only slightly modifying the way in which we support the knee of the patient; if necessary we can easily get

FIG. 17.—Tonic after-contraction of long duration in cerebral pseudobulbar paralysis.

alternately tonic and clonic after-contractions. This may be explained by first looking at the myograms. We see in both a tonic after-contraction. Now in order to get a clonus we have only to render the conditions for it as favourable as possible. If the upper part of the leg be immovably fixed the increase of tension in the quadriceps caused by the tonic after-contraction is sufficient to allow of the occurrence of a clonus. But if we allow the leg to be nearly fully extended during the after-contraction the tension does not rise so high as to permit of a clonus. The same is the case if the knee is bent much farther than is usual whilst provoking the knee-jerk.

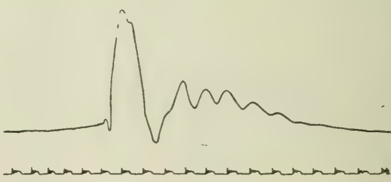

FIG. 18.—Knee-reflex, clonic after-contraction.

Muscle cut off from the central nervous system loses its tone. The normal maintenance as well as any variation of muscular tonus is a reflex action. The well-known Brondgeest experiment has probably been the first direct proof. As our knowledge of tonus is still far from complete, many have endeavoured to obtain a better insight into tone mechanism. Amongst those who have investigated the mechanical properties of normally innervated muscle substance as compared with those of muscle-protoplasm without tonus innervation, I must mention Langelaan of Amsterdam, who has contri-

buted a valuable paper on this subject in *Brain*. Professor Pekelharing in Utrecht was the first to demonstrate the chemical difference between ordinary contraction and tonus contraction. It is by the last that the creatine production is principally governed. De Boer has investigated the peripheral reflex mechanism itself and demonstrated that the centrifugal path probably lies along sympathetic nerve-fibres. Lastly, Boeke of Utrecht has found in all striped muscles new motor nerve-endings, which are not in connexion with the motor ganglion cells of the anterior grey substance in the cord. Personally I incline to the belief that Boeke's fibres, which undoubtedly are of sympathetic origin, conduct the centrifugal tonus impulses, though I am aware that Magnus and Dusser de Barenne do not share this opinion.

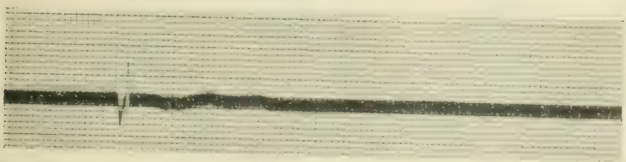

FIG. 19.—Electromyogram in knee-reflex with tonic after-contractions. Same case as fig. 17.

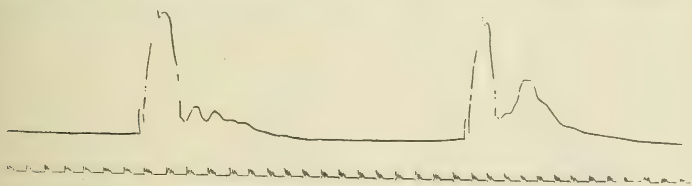

FIG. 20.—Knee-reflex with clonic and with tonic after-contraction.

The afferent reflex fibres, bearing the impulses from the muscle substance, pass through the posterior roots into the spinal cord, where they terminate in the lateral horns. From there a connector fibre passes through the anterior root, through the grey communicating rami into the sympathetic ganglion, where a third sympathetic neuron begins and returns to the motor nerve, where it runs parallel with the motor fibres to the muscle substance. Here it terminates in Boeke's end-organs. De Boer cut the grey rami communicantes in a frog and found the tonus to be diminished at the same side. A few days afterwards the muscle was stimulated by a single break shock of the induction coil. When the stimulus was applied to the spinal centre

the form of the muscle twitch was not the same as when the muscle was directly stimulated. In the former case the duration was shorter and the myogram showed the simplest and best known form. With direct stimulation the curve exhibited a secondary summit following the first one. De Boer considers this second elevation, generally known as "Funke's nose," as a tonus-contraction caused by direct

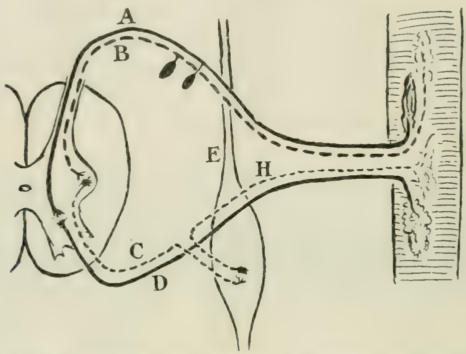

FIG. 21.—A and B, peripheral sensory neuron; C, peripheral connector neuron; D, motor neuron; H, sympathetic neuron: E, sympathetic nerve.

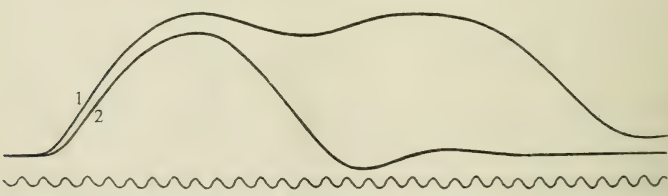

FIG. 22.—1, Contraction of gastrocnemius after direct stimulation of the muscle. 2, *Idem* after stimulation of the spinal cord, the rami communicantes having been cut five days before.

stimulation of tonus substance, at the same time as the anisotropous muscle substance is stimulated and contracts. This tonus substance can no longer be stimulated from the spinal cord after degeneration of the sympathetic fibres.

In the myogram of human muscles we practically always find the elevation of Funke; in hypertonic muscles the elevation is more plainly visible, as was clearly demonstrated by curves published by Mendelsohn

of Paris and Kollarits of Budapest. Both describe a spasmodic muscle contraction in which the tonus elevation dominates nearly the whole curve. We have seen the same thing in curves of the knee-jerks. We may therefore state that the knee-reflex is not so simple after all, but yet that it contains in itself the cause of a tonus-reflex. As this is also true of all the other deep reflexes we may say that a tendon-reflex is really composed of two superposed reflexes.

FIG. 23.—Tetani with sixteen and eighteen impulses per second ; healthy man.

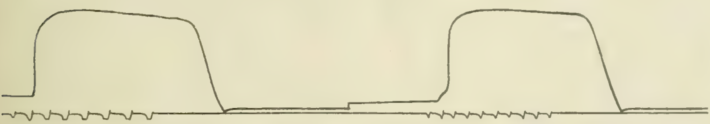

FIG. 24.—Tetani with ten and fourteen impulses per second. Disseminated sclerosis.

FIG. 25.—Tabes dorsalis. Tetani with 22 and with 24 impulses per second.

I can state here that the degree of tonus in human muscles has a considerable influence on the facility with which a complete tetanus may be provoked by faradic currents. Ratu Langi and I found that whilst in normal muscles about 18 interruptions per second of the induction coil were needed to elicit a complete tetanic contraction, hypertonic muscles gave a complete tetanus even with 10 or 12 interruptions per second. On the other hand, not less than 22 to 24 interruptions per second were needed with patients with locomotor ataxia whose muscles were quite atonic.

Let us now return to the patients with chorea minor in which the knee-reflex proper is followed by a shorter tetanic contraction. I have already remarked that this second part has a definite latency. This is

one of the reasons why I favour the opinion that we must not consider this tetanus as a voluntary contraction, but should rather regard it as a reflex contraction. This would imply that the knee-jerk has a third component—viz., a tetanic one. There are two other facts which seem to point to the same thing. In a few rare cases of tabes dorsalis or of chronic peripheral polyneuritis we find, on trying to elicit a knee-jerk, that the quadriceps contracts, but with a notably lengthened latency. This fact was observed and investigated first by Westphal, and is generally known as the pseudo-knee-jerk. Westphal regards this as a cutaneous reflex, and in some eight or ten cases in which I have seen this pseudo-patella-reflex I always found a constant though great latency of the order of 0·18 to 0·2 second. The other fact is that with many nervous persons the tapping of the patella tendon does not elicit a simple twitch but a jerk, immediately followed by convulsive extension of the whole lower limb. We can best explain these three different varieties of the knee tendon-reflex by assuming that not only a medullary centre but also a cerebral centre is provided for the knee-reflex. The reflexogenous zone for the central component extends over the part of the skin nearest to the patellar tendon. The centre itself is a secondary one of but little general use, and becomes active only under favourable circumstances, such as are found in patients suffering from chorea minor, or in highly-strung nervous persons, where either its excitability is increased or voluntary inhibition is diminished, or in rare cases of locomotor ataxia or polyneuritis, where it acts vicariously for the medullary knee-reflex centre. In character this cerebral knee-jerk is a skin-reflex with tetanic response. As the ordinary knee-jerk, which it should sustain and strengthen if necessary, is generally quite adequate and sufficient for its purpose, the action of the cerebral part of the reflex is hardly ever called for. Even when this might happen voluntary inhibition will generally prevent its activity, just as might happen in a minor degree with some ordinary inconstant skin-reflexes. Though this explanation permits of viewing a few widely different subjects from the same standpoint, I should have hesitated to accept it were it not that I had found the same thing with another group of reflexes which I described a few years ago under the name of shortening reflexes. If in some patients the relaxed foot is passively bent with a quick movement, so as to bring the ends of the tibialis anticus muscle nearer together, we sometimes observe a short contraction of this muscle. In the same way a passing contraction may be provoked in the semimembranosus, semitendinosus and biceps, if

the knee is passively bent somewhat rapidly. These contractions appear when the muscle is shortened, which proved to be the reflexogenous moment. Hence the name. The latency of these reflexes is extremely short: of the order of 0·04 second, which places them on a line with

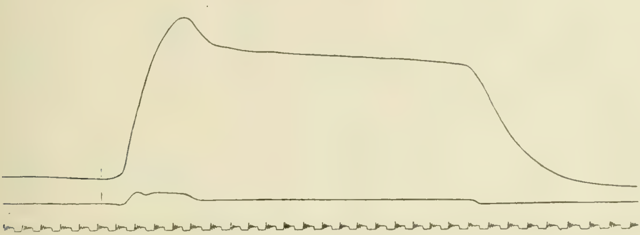

FIG. 26.—Shortening reflex in the tibialis anticus of a healthy man. Upper curve: passive displacement of the foot. Middle curve: contraction of tibialis anticus. Time marks of 0·1 second.

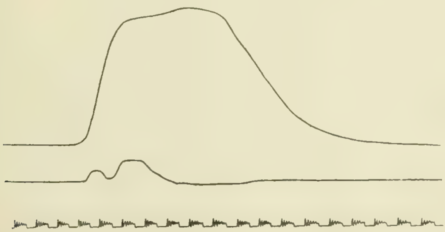

FIG. 27.—Shortening reflex of the healthy leg of a hemiplegic patient.

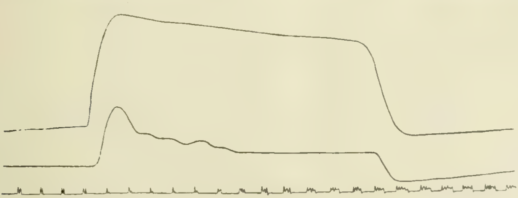

FIG. 28.—Shortening reflex of an oscillating type.

the tendon-reflexes. They also show the same peculiarities as the latter, being for instance accompanied by a tonus variation, to which the muscle twitch forms the introduction. In a few patients I was able

to record double contractions, as with the Gordon-Hey reflex. Rarely was a clonic after-contraction observed. These reflexes fail to appear whenever the tendon-reflexes are absent. But if these last are exaggerated, the same does not happen with the shortening reflexes. Curiously enough we find them generally easy to provoke in hemiplegic patients, not on the paralysed side but in the other leg. These reflexes show the same complexity as tendon-reflexes: first the tonus variation, and often the secondary tetanic component, showing them to have a cerebral centre too. If the activity of the cerebral centre be greatly increased the shortening reflex may change into Westphal's paradoxal contraction. I consider this last as a pathological variety of the shortening reflex if we except those cases in which it is of a cataleptic origin.

We have now to consider the cutaneous reflexes. These too I believe to be more complicated in the matter of the centre than we are taught in the text-books. This seems to be absolutely certain with respect to the defence reflexes from the skin, which generally possess a medullary and a cerebral centre. In animals the spinal centre is probably the more important one, whereas in man it is subordinate to the cerebral centre, which is generally located in the cortex. If this be destroyed the skin reflexes disappear, but usually reappear after a fairly long period and more often in slightly modified form or intensity. In several patients with section of the spinal cord where the post-mortem microscopical examination proved the section to be a complete one, I have seen the skin-reflexes return after an absence of six months (e.g., Brouwer). We may, and indeed ought to, assume a double centre for the skin-reflexes as well as for the deep reflexes, the difference being that in the cutaneous reflexes the more important activity belongs to the cerebral centre, whereas in the deep reflexes this is only a secondary centre of minor importance. That a tonus reflex also forms part of any skin-reflex need hardly be mentioned, as we cannot imagine a tetanic contraction without tonus reflex. This last is a positive one in the contracting muscles, a negative one in the antagonists.

In a few reflexes the complexity is still more pronounced than would appear from our discussion. The best example is perhaps the plantar reflex. In children who cannot yet stand or walk the plantar reflex, obtained by stroking the lateral side of the sole of the foot, is a pure protective reflex. The knee is bent, the foot is dorsally extended, in which also the long extensor of the great toe assists; frequently the whole leg

is drawn up. As far as the great toe, it shows the reflex type described by Babinski. But as soon as the child is able to walk a new reflex appears, in which the toes are flexed, as soon as the sole of the foot is stroked or pressed. Kalischer first pointed out that this new reflex is of great teleological importance. It causes the toes to be pressed against the floor as soon as the sole of the foot touches it, making the surface of contact as large as possible and rendering the conditions for standing or walking and for keeping the equilibrium as favourable as they can be made. Now the old extension reflex has not disappeared; it still exists but is invisible on account of the presence of the new and stronger reflex. The strength of the new reflex is probably to be found in the strength of the contracting muscles, as the toe flexors are stronger than the extensors in physiological condition. Consequently the preponderance of the action of the flexors over the extensor muscles practically renders the escape reflex unnoticeable. If the pyramidal tract has degenerated the toe flexion reflex disappears, only to leave the extension reflex of the Babinski type. How is this fact to be explained? Babinski, Kalischer and others speak of a disturbance of the physiological preponderance of the flexor muscles. But why should in pathological conditions the flexion be weaker than the extension if, normally, the extension was weaker? How can destruction of the lateral tracts cause this change? My answer to the question was that the explanation must not be looked for in the muscles themselves but in the behaviour of the different reflex-centres. Before giving a fuller exposition of my meaning, I may be allowed to say a few words on the muscle action in the Babinski reflex.

A mechanical record of the contraction of the extensor hallucis longus during a Babinski reflex shows that contraction is always a long one, somewhat resembling the slow contraction in reaction of degeneration, but mostly of still longer duration. If an electro-myogram be taken at the same time we can see better what happens. In this record on a plate moving 50 millimetres a second the electro-myogram shows an irregular alternating current lasting 0·66 second. About 0·14 second after the commencement of the action current the mechanical response begins, very gradually at first and only after 0·36 second very rapidly. The whole mechanical response takes not less than 2·7 seconds. In another record we see nearly the same thing. The time between the commencement of the electrical and the mechanical response is again about 0·12 second. The duration of the action current, or—what is the same thing—of the

purely tetanic part of the contraction, happens again to be 0·70 second, whereas the whole contraction takes over 3 seconds. In yet another electro-myogram the tetanus took only 0·11 second, though the total contraction lasted rather more than 2 seconds. These records prove

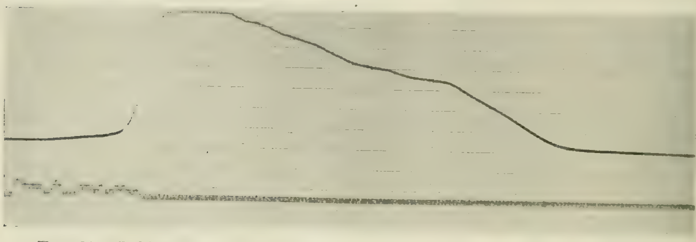

FIG. 29.—Babinski reflex. Myogram and electromyogram. 50 mm. per second.

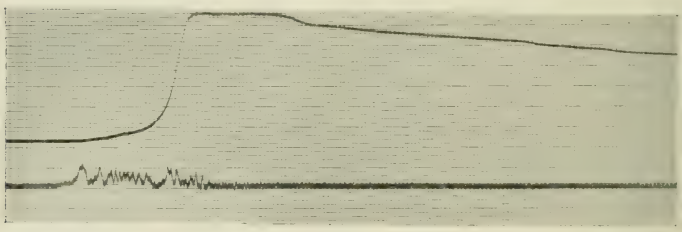

FIG. 30.—Babinski reflex. Myogram and electromyogram. 50 mm. per second.

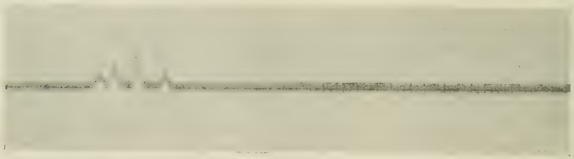

FIG. 31.—Babinski reflex. Electromyogram. 10 mm. per second.

that in the Babinski extension reflex, too, the tetanus is the beginning of the reflex which is immediately followed by a tonus-contraction. But they also show that the tetanus, which is the real primary reflex, is not sufficient in itself to cause more than a hardly perceptible movement of the hallux; it is the tonus variation that causes the

extensive movement. I need scarcely add here, that I never succeeded in getting the tonus-contraction without the tetanus. Clinical examination of the patients from whom these records were taken proved that the tonus of the muscle of the hallux during rest was not greatly changed; a passive flexion or extension of the hallux met with almost the same rather small resistance. Apparently it was not the tonus itself which was exaggerated, but the irritability of the tonus-reflex mechanism. With normal muscles we never find anything of this kind. As soon as the action current disappears the contraction ceases. Only in muscles very nearly exhausted—as we have already explained at some length when discussing the katatonus experiments—do we get after-contractions of tonic origin. Consequently there can hardly be any doubt about the interpretation of these curves.

Before returning to the question of the plantar reflex, I must point out that the centres of the phylogenetically old reflexes are situated in the spinal cord. The younger a reflex is phylogenetically, the higher up in the cerebro-spinal system must we look for its centre. Where two or more centres are present we may expect that the oldest centre will be found in the lower part of the central nervous system, generally in the spinal cord.

Let us now return to the plantar reflex. The reflexogenous zone is the sole of the foot. As part of the integument it possesses in the first place the property, belonging to any part of the skin, of being the reflexogenous zone for a protective reflex. This is a phylogenetically very early reflex with a low spinal centre. In man this spinal reflex is not adequate and it has to be assisted and restrained by cerebral impulses—involuntary as well as conscious ones—undoubtedly in a far higher degree than in animals. But this means the development of a cerebral centre which gains in importance the higher up we look in the animal series, till we arrive at man, where it has attained far greater importance than the original spinal part of the reflex.

The sole of the foot is at the same time the reflexogenous zone for the toe-flexion movement, the reflex needed for walking and standing. This reflex develops only as the child learns to stand and walk, and we may even say that the act of learning to stand is the same thing as the gradual acquisition of the reflex. As standing and walking only take place after a cerebral impulse and are only possible by continuous cerebral impulses, the whole mechanism of these actions comes very near to being a complete cerebral automatism. As soon as the cerebrum directs the foot to touch the floor toe-flexion appears as

the consequence of a phylogenetically late and consequently cerebral reflex. Now we also see why teleologically the toe-flexion reflex should be stronger than the protective toe-extension reflex in normal man. If this were not so, walking would be extremely difficult, not to say impossible, as with each step the toes would be lifted from the floor instead of being spread so as to enlarge our contact with the earth. That is the reason why the act of learning to walk is largely synonymous with the acquisition of this reflex. Even the age of the child is immaterial; in rare cases where children learn to walk at a more advanced age, as in late rickets, the original Babinski type of the plantar reflex persists till the child is able to walk. But as soon as this happens the flexion type of the plantar-reflex is provoked by touching the floor with the sole of the foot as well as by striking it.

Starting from these considerations we arrive at a plausible explanation of the Babinski reflex in lateral sclerosis. As the centrifugal impulses for the plantar reflex pass through the pyramidal tract, the cerebral centre is put out of action. But the spinal centre and its efferent and afferent paths remain intact, certainly for the toe-extension reflex, and if a spinal centre for toe-flexion exists, for this centre also. The activity of the spinal toe-flexion centre, which is a phylogenetically late reflex, need scarcely be considered as compared with the significance of the centre for the extension of the hallux, which is a very early reflex in phylogenesis. And though even the activity of this last centre is relatively small as compared with its own cerebral component, it is readily redeveloped. Consequently toe-extension reappears, though not in a very pronounced form. As at the same time the secondary tonus reflex is greatly increased owing to the enhanced irritability of its spinal centre, which is no longer controlled by the higher tonus-centres, we finally get an ample dorsal extension of the toe.

Summary.

In this lecture I have tried to demonstrate the advantage to be derived from the use of the string galvanometer in clinical neurological research, where it can differentiate the several modes of muscle contraction. Graphic records of the electrical and mechanical responses in human muscles show both the innervation and the mode of contraction to be a double one. We have to distinguish between a tetanic and a

tonic innervation as well as between contractions of tetanic and tonic character.

The graphic study of reflexes proves them to be much more complicated than is generally understood to be the case. The complication arises from the fact that every simple or tetanic reflex is in itself the cause for a secondary tonic reflex. Moreover most reflexes are governed by more than one centre. Application of these principles has been made to explain the genesis of the Babinski form of the plantar reflex.

Proceedings of the Section of Neurology of the Royal Society of Medicine.

President—Dr. ERNEST S. REYNOLDS.

In the Chair—Dr. W. ALDREN TURNER, C.B.

Meeting held October 7, 1920.

Aphasia: An Historical Review.

(THE HUGHLINGS JACKSON LECTURE FOR 1920.)

By HENRY HEAD, M.D., F.R.S.

THE evolution of our knowledge of cerebral localization is one of the most astonishing stories in the history of medicine. Throughout the Middle Ages the brain was supposed to contain three ventricles, each of which was the dwelling place of one or more aspects of the psyche [19]. The anterior chamber received the nerves of taste, smell, sight and hearing, and was the seat of the "sensus communis"; in the middle ventricle dwelt the faculty of cogitation and reasoning, whilst the posterior one was the seat of memory.

Under the profound influence of Descartes and his followers the vivid intellectual life of the seventeenth and eighteenth centuries swept away all traces of this scholastic materialism. Many guesses were made, but no satisfactory theory was evolved to account for the relation of mind and body; even as late as 1798, Soemmering thought the seat of the soul was in the fluid which filled the ventricles. No one presumed to imagine that the activities of the mind bore any direct relation to the life of the brain. Gall first suggested that this apparently uniform mass was made up of organs which subserved the manifestations of the vital and moral faculties of man. These could be divided into three groups: (1) Those concerned with the exercise of "vital force," which he located in the upper part of the spinal cord and medulla oblongata; (2) the "inclinations and affections of the soul," situated in the basal

portions of the brain, and (3) the "intellectual qualities of the mind," associated with the cerebral hemispheres.

We habitually think of Gall with derision, as a quack who was responsible for phrenology and the study of bumps on the head. But it is to this man that we are really indebted for the beginning of the conceptions we now hold of the relations of the various portions of the nervous system to one another.

Franz Joseph Gall was born on March 9, 1758, at Tiefenbronn, in Baden. He studied medicine in Strasbourg, and in 1781 settled in Vienna. From 1796 to the end of 1801 he lectured on a new theory of the functions of the brain. His views were considered so subversive of religion and morals, that on December 24, 1801, his lectures were forbidden by an autograph letter of the Kaiser. Gall seems to have remained in Vienna for the next three years, and then journeyed through Europe with his pupil Spurzheim, lecturing as he went [10]. In 1807 he reached Paris, and in the following year presented a memoir to the Institute of France, entitled "Introduction au Cours de Physiologie du Cerveau." On this treatise a committee specially appointed drew up an unfavourable report. Gall and Spurzheim in 1809 published their "Recherches sur le système nerveux en général et sur celui du cerveau en particulier," which comprised a categorical examination and reply to the objections of the committee's report [11]. This was their first serious publication in French, and it contains all the views put forward by Gall at one time or another. Subsequently he issued in Paris a full account of his work in eight volumes, with an atlas of a hundred plates [12].

The doctrines of Gall had an enormous influence upon the thought of his time ; but the only part of his theory which we attribute to him in the present day is the idea that the underlying structure of the brain has an effect upon the formation of the skull, and in consequence that character could be foretold from the external conformation of the head. But in reality this is but a small part of the theory put forward by Gall. He was the first to insist that the brain should be examined anatomically from the spinal cord upwards ; for he laid down that the functions of the brain could only be explained by considering their relations to those of the spinal cord. All nerve fibres ended in grey matter, and the white matter of the nervous system served for conduction only. He pointed out the analogy between the grey matter which covers the hemispheres of the brain and of the cerebellum, with that which forms the corpora quadrigemina and optic thalamus. All

nerves, whether they belonged to the brain or to the spinal cord, took their origin in grey matter, and the substance of the cerebrum and cerebellum could not be considered to be in direct connection with the peripheral nerves.

These ideas have been accepted into the body of general knowledge, but they were accompanied in Gall's exposition by the most fantastic theories with regard to the "moral" qualities. He pointed out that injuries to the head showed that the mind was associated with the brain, and that the brain serves as the organ of intellectual life. The nervous system of man was therefore the physical instrument of his moral life, for the nerves were the conductors by which the will was transmitted to all parts of the body. Then he set about to consider in what parts of the brain were situated the various moral and intellectual qualities of man. At that time it was supposed that man was endowed with certain "faculties," and it was these that Gall attempted to localize in different parts of the brain. He found the organ of the generative force in the cerebellum, and those of the five senses over the front portion of the brain. There were six different forms of memory, each of which he localized in a different "organ," or as we should say "centre." There was the memory of things, the memory of locality, the name-memory, the verbal and grammatical memory, and the memory for numbers. All of these were situated in those portions of the brain which lay in relation with the posterior part of the orbital cavity. It would seem that this localization dated back to Gall's earliest years; for as a boy he noticed that his companions who had "des yeux pochetés" (prominent eyes) had a gift for languages and a memory for words. He therefore concluded that the parts of the brain which pushed the eyes forward must be associated with the different varieties of verbal memory. Translated into anatomical terms, this meant that the frontal lobes and particularly their under surface must be devoted to these forms of memory.

He appears to have taken no precautions to verify this localization, but he had the opportunity of seeing an officer in whom a foil had passed through the base of the frontal lobes, producing grave disorders of speech. He gives a very short description of this case, but seems to look upon it simply as confirmatory evidence of his theory.

Gall died on August 22, 1828, and gradually most of his views passed into oblivion or came to be treated with derision; but his conception that speech and the memory for words were situated in the frontal lobes still found supporters. Its warmest champion was

Bouillaud, who was appointed Professor of Clinical Medicine at the "Charité" in 1831, and at the time that Broca made his first post-mortem was 65 years old, and the "doyen" of the faculty. He was an ardent follower of Gall, and offered a sum of money to anyone who would produce the brain of an individual who had lost his speech in which the anterior lobes presented no lesion.

In 1861 Broca had just become the head of the Surgical Unit at Bicêtre. An elderly man was admitted to his wards (aged 51) with a diffuse gangrenous inflammation of the right lower extremity. He was completely aphasic, and died a few days later; Broca demonstrated the brain at the Société d'Anthropologie, of which he was the secretary [5]. This brain showed destruction of the posterior part of the second and third frontal convolution, the inferior portion of the Rolandic area and the first temporo-sphenoidal convolution. It was preserved in the Musée Dupuytren, and was subsequently examined more carefully by Pierre Marie [16, No. 3], who has pointed out that the lesion also occupied the supramarginal gyrus, a part which at that time was not considered of any importance from the point of view of speech. This was followed by a second case [7] in which the situation and nature of the lesion was acknowledged to be more doubtful (November, 1861). This brain also has been examined by Pierre Marie, who could find no lesion of the third frontal convolution; but there seems to have been some wasting at the foot of the second frontal convolution, which is not uncommon in a senile brain. Broca himself says that this lesion is incomparably more circumscribed than that of the first brain, but he continues, "In comparing the two specimens it is possible to make certain that the centre of the lesion is identically the same in the two cases."

As may easily be imagined, these communications led to the greatest excitement in the medical world of Paris. Bouillaud, and his son-in-law Auburtin, greeted Broca as a convert to the doctrines of Gall. The localization of speech became a political question; the older conservative school clung to the conception that the brain "acted as a whole," whereas the younger liberals and republicans passionately favoured the localization of function. For the next few years, every scientist of importance in Paris took one side or the other. Trousseau brought the weight of his authority into the scale on the side of Broca, and invented the word "aphasia" to replace Broca's "aphemia." This was an unjustifiable change in nomenclature, which as Broca complained "brought back into pathology the very confusion I thought to dissipate."

Broca described aphemia as follows: "There are cases where the general faculty of language persists without alteration, where the auditory apparatus is intact, where all the muscles, without excepting those of the voice and articulation, obey the will, and where in spite of this a cerebral lesion abolishes articulate language; this abolition of speech in individuals who are not paralysed or idiots constitutes a symptom sufficiently singular to be given a special name. I shall give it the name of 'aphemia' because what is wanting in these patients is solely the faculty of articulation. They hear, they understand all that one says to them; they have their intelligence, they can emit vocal sounds with ease; they can execute with the tongue and cheeks movements much more energetic than those required for the articulation of sounds. Moreover, the answers they can make are reduced to a very few articulated sounds, always the same and always disposed in the same manner. Their vocabulary is composed of a short series of syllables, sometimes of a monosyllable which expresses everything, or rather it does not express anything, because the single word is often foreign to all vocabularies. Some patients have not even this vestige of articulated language; they make vain efforts without pronouncing a syllable" [6].

Broca had discovered a definite clinical condition, which he called "aphemia," produced by destruction of the substance of the brain, and he believed that he had found the seat of the faculty of articulated language in the third frontal convolution.

Interest in the question of localization of speech rapidly spread to England, and in 1864 Hughlings Jackson published his first paper on "Loss of Speech with Hemiplegia of the Left Side" [14, No. 2]. This was followed by a series of contributions on the subject in which many other physicians joined.

In 1868, the British Association for the Advancement of Science held its Annual Meeting at Norwich, and M. Broca opened a discussion on aphasia. He was followed by Dr. Jackson. No record of these contributions is to be found in the *Proceedings of the British Association*, but Broca's paper was published in the *Tribune Médical* [8].[1] An abstract of Jackson's paper appeared in the *Medical Times and Gazette* for September 5, 1868. This has been reprinted amongst the collected papers in *Brain* [14, No. 17].

[1] I was unable to find this paper in London and owe my knowledge of its contents to the kindness of Professor Pierre Marie, who caused a copy to be made by hand. This he presented to me in memory of his master, Broca, for whom he acted as Interne in 1880.

Thus, we are able to compare the views of the French and English protagonists of localization, and it is no wonder that at that period, and, under those conditions, Jackson's views produced little serious effect. Broca is clear, definite and precise, and I shall therefore begin this stage of the history by summarizing his views.

He divides affections of speech of central origin into two main groups —"Aphémie" and "Amnésie verbale," and lays down clearly the difference between them. The aphemic patient has a profoundly reduced vocabulary and may be speechless, except for some monosyllables, oaths and words that do not seem to belong to any language. His ideas are intact, as shown by gestures, and he can understand what is said to him and recognizes words and phrases which he cannot pronounce or even repeat. On the other hand, the amnesic patient no longer recognizes the conventional associations established between ideas and words. He can pronounce them, but they do not seem to have any bearing on the ideas he wishes to express. He is able to show by gestures that he has not lost all kinds of memory, but it is the special memory, not only of spoken but of written words, that is lost.

So far Broca's statements are admirably clear and consonant with fact. When however he tries to determine the site of the lesions responsible for these conditions, he becomes confused and indefinite. He saw clearly that the larger number of cases did not correspond to these two clean-cut divisions. In practice, both states can exist together and this greatly hampered the determination of the position of the lesion. He utters a wise warning on the necessity for drawing conclusions from selected cases only, and insists that it is "aphemia" which is caused by a lesion of the third frontal convolution.

Let us now turn to Hughlings Jackson's contribution to this discussion. He said nothing about localization beyond referring to his previous papers. As far as can be gathered from the summary [14, p. 59], he confined his attention to the nature of the loss of speech produced by cerebral lesions. He pointed out that healthy language consisted of two inseparable, yet distinct, forms: (1) Intellectual—i.e., the power to convey propositions; (2) emotional—i.e., the ability to exhibit states of feeling. These two are separated by disease. It is intellectual language alone which suffers in most of these cases; emotional language usually escapes altogether. Cases of affection of speech can be divided into two groups, in the first of which the patient is speechless, or nearly so; whilst in the second, he has plenty of words but uses them wrongly. He denied that there was such a thing as a

"faculty of speech" or a "faculty of memory," and says "The question is not, how is general mind damaged, but what aspect of mind is damaged?"

Thus, he laid down in this discussion the fundamental basis of the whole of his future treatment of the subject. It is the power to form propositions that is affected, and not the memory of words or faculty of language. The higher the propositional value of the task the patient is asked to carry out, the less he will be able to respond; whereas, the lower or more automatic the action required, the easier will it be to perform.

But subsequent investigators took no care to emulate the clinical acumen of Broca or the psychological insight of Hughlings Jackson. Bastian published his well-known paper in 1869 which had such an evil influence on the subsequent course of the discussion. He started from the *a priori* assumption that we think in words, and that words are revived in the cerebral hemispheres as remembered sounds. He talked of lesions of special fibres and centres, and set the points on the catastrophic road to schemas and diagrams. His subsequent Lettsomian Lectures [3], re-published in 1898 under the title "Aphasia and other Speech Defects," had, as a frontispiece, the famous diagram so often cited in subsequent discussions [4]. This book is little more than a widely expanded version of his original paper, and by its simplicity and dogmatism seduced the younger generation away from the difficulties and complexities of Jackson's doctrine.

The first actual diagram was published by Baginsky in 1871 [1], who like most of the Germans was unaware that Bastian had already described word deafness. From this time onwards the rage for diagrams became a veritable mania. No one could write on aphasia without producing a new diagram of centres and the paths between them. Each author twisted the clinical facts to suit the lesions he had deduced from his pet schema. Wernicke in 1874 [21] localized the centre for auditory images in the first left temporal convolution, but would allow no centre for reading and writing; oral speech alone possessed cortical centres, one of which was guiding and the other emissive. Between these two lay the island of Reil, and three main forms of aphasia must therefore be possible. The first of these was sensory, and the lesion was situated in the temporal convolution. The second was motor and the lesion lay in the third frontal convolution. The third variety was due to interference with conduction between these two centres.

How far the writers of this period were compelled to lop and twist the clinical facts to fit the procrustian bed of their hypothetical conceptions is shown by the famous case published by Wernicke in 1903 as "A Case of Isolated Agraphia" [22]. Now, such a title can only mean that in this patient every other act of language could be perfectly performed, except that of writing. But when the record of the case is examined, the patient appears to have had much difficulty with spontaneous speech, and in understanding what was read. She could not comprehend many spoken words, nor could she carry out commands given verbally. She is said to have shown almost complete inability to write, and it was with great difficulty she could be brought to make the attempt. She was also unable to draw to command, although she could copy drawings and writing, and she failed to say the days of the week or the months in their proper order. In the solemn discussion which follows, on the nature of disorders of speech, it is difficult to decide whether the clinical obtuseness or want of theoretical insight is more worthy of wonder.

Amongst the various diagrams that of Lichtheim [15] attained the greatest popularity. It was definite and precise; every form of aphasia was accounted for by a lesion of some hypothetical centre or purely imaginary path. Teachers of medicine could assume an easy dogmatism at the bedside and candidates for examination rejoiced in so perfect a clue to all their difficulties. But serious students could not fit these conceptions of aphasia with the clinical manifestations; incredulous of such scholastic interpretations, they lost interest in a problem of so little practical importance.

In 1906, Pierre Marie startled the medical world by three papers in the *Semaine Médicale* on "Revision of the Question of Aphasia" [16]. The first of these bore the aggressive title, "The third left frontal convolution does not play any special rôle in the function of language," whilst the third was a fascinating historical essay on "Broca and his times," together with an account of the re-examination of the hemispheres of his two first patients. This was followed by a series of papers on the subject of aphasia and its cerebral localization, culminating in the monograph of Moutier which appeared in 1908 from the laboratory of Pierre Marie at Bicêtre [17].

Marie laid down that the aphasia of Broca was not a clinical entity but a "syndrome." It is a combination of two distinct troubles which he called "anarthria" and "aphasia." By "anarthria" he understood simply the inability or difficulty of articulating words. On the other

hand, aphasia was the sensory aphasia of Wernicke, characterized by troubles of internal language, which showed themselves directly in alterations of speech, reading and writing. There is nothing of aphasia in the motor trouble of the anarthric. He understands, he reads and he writes. His power of thinking is intact, and expression is possible by every other means except words, internal language remaining unaltered. There is only one aphasia, the aphasia of Wernicke, and the term "sensory aphasia" must disappear.

But the greatest excitement was produced by Marie's views on the cerebral localization of the lesion responsible for these defects of language. Of the two factors which constituted the aphasia of Broca, the anarthric aspect was due to a lesion of the "lenticular zone" whilst the aphasic side was produced by one in "Wernicke's zone." This latter was not a special loss of word memories, but was a defect of general intelligence, and of special intelligence of language.

In Moutier's monograph, "L'Aphasie de Broca," he gives an interesting history of our knowledge of aphasia, followed by a reconstruction of the nature and symptoms of this disorder from the point of view of his master, Pierre Marie. One of the most valuable portions of this book consists in a series of reports, both clinical and pathological, of a large number of cases. The second part is prefaced by a scheme of examination which is singularly complete.

By this time the differences of opinion in Paris had become so acute, that three sittings of the Société de Neurologie de Paris (June 11, July 9, and July 23, 1908) were devoted to a discussion of this question [9]. Unfortunately, though an attempt was made to keep the clinical and pathological aspects apart, this was impossible in practice. Two questionnaires were prepared to serve as the basis for discussion. The first of these was divided into "Étude anatomique" and "Étude clinique," whilst questionnaire B consisted of three portions: (1) "Clinique"; (2) "Anatomie normale et pathologique"; (3) "Physiologie pathologique." The second of these was accepted as the basis of discussion, which began as follows: "Are motor and sensory aphasia clinically different from one another, or is motor aphasia sensory aphasia accompanied by anarthria? If there are differences, what are they?" Unfortunately this led to a discussion based entirely on terms such as "Broca's aphasia," "total aphasia," "word blindness," "word deafness," "alexia," "agraphia," &c. Marie held strictly to the views set out above, whilst Dejerine defined his position as follows: "The aphasia of Broca is a motor aphasia and total aphasia is both sensory

and motor. The aphasia of Broca is accompanied by a loss of words, marked trouble in writing, some alexia, though not excessive; the patient is able to transcribe print into handwriting. On the other hand the sensory element consists in word deafness and word blindness.

Dejerine attacked the use of the word "anarthria," which should, he contended, be kept for definite troubles of articulation. Marie represented that he had chosen this word because cases of mere motor aphasia showed no disturbance of internal speech.

Dupré and others insisted that the motor aphasic had difficulty in evoking his words. Aphasia is a psychical defect and trouble of language ("langage"); anarthria, on the other hand, is a motor defect and a trouble of speech ("parole"). Marie answered that the anarthric had no trouble in evoking a word, but solely in pronouncing it, and he agreed to substitute the word aphemia for anarthria, provided the meeting would acknowledge that the phenomenon was not associated with a defect of internal speech.

The next sitting and the first half of the final sitting were devoted to an attempt to settle the situation of the lesion responsible for the various forms of aphasia. But, as no satisfactory clinical definition had been arrived at during the earlier phase of the discussion, the greater part of the time was spent in discussing the limits of Marie's lenticular zone and its justification. Finally, the meeting turned to a consideration as to whether there were disturbances of intelligence in motor aphasia, and if so, what form they took, and secondly, whether intelligence was disturbed in sensory aphasia. At this stage, Marie brought forward his views on the evident diminution of general intellectual capacity, which is found in those cases which he describes as the "aphasia of Broca." This is in no sense a dementia; he insisted on the exactness of the terms that he had used. "Il y a une diminution très marquée dans la capacité intellectuelle en générale" (p. 1037). This intellectual disturbance is a specialized defect. He admitted that the "zone of Wernicke" is the part where a lesion produced with certainty troubles of language, but he denied independent existence of special sensory word centres which had been attributed to this portion of the brain.

Marie fought hard for simplification and made a bold attempt to sweep away the vast accretion which had obscured the question of aphasia; but no one ventured to suggest that the greater part of the discussion of the clinical manifestations was purely a verbal battle. Had the disputants been familiar with the work of Hughlings Jackson, they would have recognized that, from the point of view of the question

at issue, all these terms such as "aphasia of Broca" "total aphasia," "word blindness," and "word deafness," were pure phrases, and did not in reality correspond exactly to any clinical phenomena.

I have kept the views of Hughlings Jackson until the last, because his attitude towards the question was entirely foreign to any conception put forward in the history of aphasia. His views bore no fruit during his lifetime for two reasons. Firstly, they were published in a series of papers between 1864 and 1893, many of which were inaccessible on the Continent. Secondly, the style in which they were written makes them peculiarly difficult to read. He was so anxious not to overstate his case that almost every page is peppered with explanatory phrases and footnotes, so that the generalization can scarcely be distinguished from its qualifications. English students, accustomed to the fluent dogmatism of his contemporaries, turned away from the bristling difficulties of Hughlings Jackson's papers. Finally, the ideas he propounded were entirely opposed in their nature to the current opinions of the day. No one assimilated his views on defects of function and applied them to actual cases of loss of speech. We failed to appreciate how much closer these conceptions would have led us to the phenomena of aphasia than the glib generalities founded on the supposed anatomical facts of cortical localization.

When however I became interested in working out the problem of aphasia in the light of our researches on the effects produced by cortical lesions on sensation, it became obvious that none of the current doctrines corresponded to the clinical facts. I set to work to make a collection of Jackson's papers on the subject, and was startled to find that he had long ago arrived at several of the general conclusions which had led me to disbelieve the teaching of my contemporaries. In 1914, Pick's remarkable book, "Die agrammatischen Sprachstörungen" [18] fell into my hands and determined me to republish Jackson's papers on aphasia in *Brain* [14].

In spite of its occasional obscurity Jackson's work is of peculiar importance to the neurologist of to-day, both as a practical guide to the clinical phenomena of aphasia and an understanding of the processes which underlie the production of speech. His views can be summed up shortly as follows:—

(1) In 1868, he pointed out that patients with aphasia can be divided into two main groups. In Class 1 the patient was almost speechless or speech was gravely damaged, but in the worst of these cases the patient

could utter some one or two unvarying words or jargon. Class 2 comprises those who have plentiful words but habitually use them wrongly.

(2) The loss of power to carry out an order depends on the complexity of the task. The more abstract the conception, the more likely is the patient to fail in executing it, although he may succeed when it is put before him in a simpler and more descriptive form. Thus, the patient who could not find the word "kitten" called it "a little fur child," and one who could not draw a square when asked to do so, drew a perfect square when told to draw a block of wood. Thus, it is most important, when asking an aphasic patient to carry out some order, to present it to him if possible in several forms, noting his response in each case; because a man cannot write the alphabet, we must not assume that he cannot write a letter.

(3) The higher and more voluntary aspects of speech tend to suffer more than the lower or automatic. The least voluntary speech is that which is emitted under the effect of emotion, such as exclamations, oaths, and words such as "good-bye," rising to utterance under the impulse of a moment. In many cases "yes" and "no" can be used approximately as propositions, and even words and phrases of true propositional value may spring to the lips of the aphasic patient. But in such cases he is usually unable to repeat at will the phrase he has just used under an appropriate impulse. The "speechless man is not wordless," and the apparent inconsequence of observations on persons with aphasia is to a great extent removed by an analysis of the conceptual value of the words and phrases which are actually uttered.

(4) Writing is affected, not as a separate "faculty" but as a part of the failure to propositionize in words. There is no such condition as pure "agraphia." A man who cannot write spontaneously may be able to copy printed matter in perfect handwriting. The "faculty" of relating handwriting to print is intact; he cannot write voluntarily because he has lost the use of written words in propositions. Hence, he can usually write his name and address, because in most of us this has reached with time more nearly the level of an automatic act.

(5) Patients with such affections of speech may not be able to read aloud or to themselves, when asked to do so; but they can understand what is read to them and may even obey written commands, although unable to reproduce them in words. This is not due to some loss of function called "alexia," but to an inability to reproduce a proposition which, on the other hand, they may be capable of receiving accurately.

(6) "Imperception" is on the receptive side what aphasia is in the

"word series." In many cases the two conditions are combined; but they may exist separately, and where aphasia is present without "imperception," images remain intact. Thus the patient may be able to point to colours and objects when they are named; he continues able to play cards or dominoes; he recognizes handwriting, although he cannot read the words written; he knows poetry from prose by the different endings of the lines on the right of the page.

Thus, affections of speech are caused (*a*) on the emissive side by inability to form or to express a proposition in words; (*b*) on the receptive side by failure of those mental processes which underlie perceptual recognition.

(7) External and internal speech are identical, except that one leads to the utterance of articulated words, whilst the condition of internal speech can be discovered by writing only.

(8) Behind external and internal speech stands the proposition which, when verbalized, can be expressed in speech or writing. This proposition is necessary for clear and logical thought, but not for all thinking. When this aspect of speech is affected, the patient cannot retain a sequence of abstract propositions, because he is unable to formulate them at will to himself. He can think, but he is "lame in thinking."

(9) If, however, "imperception" is added to such defect of speech the patient will not only suffer from "inferior speech," but will show signs of "inferior comprehension."

(10) In the majority of cases of affections of speech mental images are unaffected. This extremely important contribution to the theory of aphasia has been entirely neglected by neurologists. For almost every hypothesis propounded in the last forty years presupposes some defect in "auditory" or "visual word images."

In 1910, it became obvious to me in the light of our work on the part played by the cortex in sensation, that new tests must be devised before the clinical investigation of aphasia could produce any satisfactory results. It is not sufficient to hold up some object and ask the patient to name it; at one time he may be able to do so, at another he fails completely. No conclusion can be drawn from one or two questions put in this way. His power of responding must be tested by a series of observations in which the same task occurs on two or more occasions.

Not only is it necessary to arrange the tests in sequence, but each set must be put before the patient in several different ways. For example, six common objects are laid on the table in front of him, and

he is asked to point to the one which corresponds to a duplicate placed in his hand out of sight. This is repeated for from eighteen to twenty-four observations, so that the choice of any one object recurs three or four times in the course of the series. Then he indicates each one in turn as it is named by the examiner, or makes his selection in answer to printed words set before him on a card. He next gives names to the various objects, one by one, and finally writes them down without saying anything aloud. The order in which these various acts are performed remains the same throughout the different aspects of the test. This alone makes it possible to draw any conclusion from the inconstant responses, which are so disconcerting, unless the answers are recorded in this manner. Moreover, this method enables us to learn how the patient responds to the same series of tests put before him in different ways. I have described all these methods of examination in a recent number of *Brain*, and shall not dwell further upon them [13]. The principle upon which they are based is to set a similar task before the patient through various forms of command; these he must execute by different methods. He is no longer asked to speak, to read or to write ; but all these methods of expression are used to answer the same series of tests.

As soon as I began to apply these methods of examination to patients suffering from aphasia, it became obvious that none of the ordinary theories bore any relation to the facts I discovered ; it was not until I had read through a complete series of Jackson's papers that I recognized the importance of the principles he had enunciated in the light of my own observations.

Shortly after the beginning of the War, patients began to pass into my care with wounds of the head, who suffered from defects of speech slighter and more specialized than any I had seen before. Moreover, these young men were struck down in the full pride of health; many of them were extremely intelligent and anxious to be examined thoroughly. As their wounds healed, they were encouraged and cheered by the obvious improvement in their condition. They were euphoric rather than depressed, and in every way contrasted profoundly with the state of the aphasic patient met with in civilian practice. From this time onwards the work progressed rapidly, and the results that I shall lay before you are based mainly upon these cases of unilateral wounds of the head.

But, before we enter on the positive results of these researches, I am anxious to clear the ground of certain preconceptions. We know

that speech can be affected by destruction of the substance of the brain, but this does not show that "the faculty of speech" is localized in any area of the cortex. We should as soon expect a special centre for eating as for speech; both are complex acts which do not correspond to any specific group of functions. No lesion, however local, can affect speech and speech only. The cerebral injury disturbs certain physiological processes which subserve the complex acts which we speak of as speech. Now, no anatomical lesion corresponds exactly to a single group of physiological functions; the acuteness and severity of the onset is often a more important factor in determining their nature than the extent of the lesion. On the other hand, there is no single psychological function or "faculty" corresponding to speech; any organic injury which produces a disorder of speech disturbs other functions not usually associated with language, or leaves unaffected much that undoubtedly belongs to speech. We must therefore get rid of all those *a priori* conceptions, which underlie such terms as "motor" and "sensory" aphasia, "alexia," "agraphia," or "amnesia verbalis." No disorder of speech due to a unilateral lesion of the brain corresponds exclusively to any one of these categories, still less can these hypothetical conditions be associated with limited destruction of any one part of the brain.

An organic lesion disturbs certain physiological processes which are necessary for the complex acts which underlie the use of language. Words, numbers, pictures, and every function which depends upon the use of these symbols in constructive thought may be affected. Any mental process is liable to suffer which demands for its performance exact comprehension, voluntary recall, and perfect expression of symbolic representations. I have therefore suggested that the various functions disturbed in aphasia and allied conditions might be spoken of as "symbolic thinking and expression," because it is mainly the use of words, numbers and pictures which suffers in these disordered states. But this name must not be taken as in any way defining the group of processes affected; I should have preferred, had it been possible, to have employed some entirely indifferent term. For it is symbols used in a particular manner which suffer in these disorders and not all symbolic representations.

The more nearly the symbolic action approximates to a perfect proposition, the greater difficulty will it present, and the patient will probably fail to execute it correctly. Conversely, the more closely it corresponds to matching two sensory patterns, the less likely is it

to be disturbed. Highly complex symbolic acts suffer more gravely than those of lower propositional value. For example, if a patient seated opposite the observer attempts to imitate a series of movements which consist in touching the eye or ear with one or other hand, he may fail grossly. He can however execute them perfectly when the observer stands behind him and they are reflected in a mirror. For in order to imitate movements sitting face to face he must formulate the various factors which make up the command; whilst to copy them when reflected in a mirror is an act of almost pure imitation. But if, instead of imitating the movements, he is asked to write down what he sees in the mirror, he again fails grossly; for he is now compelled to formulate the task in words, and so fails to describe gestures he can imitate perfectly.

The higher the propositional value of the mental act, the greater difficulty will it present. Thus, the patient may be able to execute a printed command to hold up his hand, although he is unable to carry out an order to touch with it his eye or his ear. The addition of the second factor has rendered the task too difficult. The larger the number of alternatives presented by the order, the more certainly will the desired action be defective. All propositions express an abstract relation; but even the same generalized statement may vary in difficulty according to the means by which it is expressed. For example, during the compass test, the aphasic patient may be entirely unable to record in speech, or in writing, whether he has been touched with one or two points; but if the figures 1 and 2 are written on a sheet of paper, he can indicate correctly whether the contact was single or double. Under the ordinary conditions of this test, he is not only compelled to formulate in words his sensory impressions, but he must express them in verbal symbols; but when it is carried out according to the second method, he has only to determine whether his sensations are of one or two points, and the means of expression lie on the table before him. When the defect consists of want of power to evoke words or names, the patient may be unable to name a set of common objects, although he can choose them correctly to oral or printed command. The verbal symbol conveys its proper meaning when presented ready formed in sound or in print; but to produce it freely at will demands a greater perfection of symbolic thinking and expression. In the same way when an aphasic calls scissors "that is the tweezers to cut with," he is expressing a lower grade of symbolic recognition. One of my patients was unable to name a series of colours placed before him, and could not choose them cor-

rectly to oral or printed commands. But if instead of the names he was allowed to call black, "what you do for the dead"; red, "what the staff wear," with similar descriptive phrases for each of the other colours, he named them all correctly. These metaphorical modes of expression are simpler than names which consist of a single word; for the more concrete is lower in the hierarchy of mental processes than the general and abstract.

Behind every propositional expression lies recognition of the meaning of symbols; I must be able to appreciate the full significance of words, numbers, and pictures before I can pose them in propositional form. This loss of meaning is the cause of many aphasic defects; thus, the short and the long hands of the clock have acquired a significance which converts each one of them into a direct symbol, and they are often confused, or used wrongly in cases of aphasia.

When adding two numbers together, it is easier to reach the answer by counting than by direct proposition. Thus, many aphasics can add 6 and 3 by saying to themselves 6, 7, 8, 9, but cannot formulate $6 + 3 = 9$. For the same reason some of them could set the clock correctly to a command given in railway time, but not if it was expressed in words. Told to set the clock at 4.45, the patient placed the hour hand at 4, and then swept the other round the face to a position he associated with 45 minutes. But asked to set a quarter to five, he placed the one hand at 5, whilst the other hovered uncertainly between a quarter past and a quarter to. This does not depend on a difference in the power of comprehending words and numbers; for when the patient attempted to tell the time it was equally difficult for him to evoke the names "4.45" and "a quarter to five."

Many of the disorders found in aphasic patients are the result of inability to recognize the significance of words and names. If, however, they fail to appreciate with certainty the ultimate meaning in thought, they are liable to become confused over the intention of an action, whether self-suggested or imposed from without. The patient no longer perceives clearly the goal at which he must aim. Thus, a young officer could thread a quadrilateral frame for his bee-hives if the action consisted in bringing the wire across from one side to the other and back again through neighbouring holes, but as soon as he attempted to go from corner to corner, he failed entirely.

Perfect symbolic thinking and expression demand that words, numbers, pictures, and all they stand for in thought should be susceptible of voluntary manipulation. It is this aspect that is usually

affected in the more verbal forms of aphasia and in those that are characterized by the use of jargon; these are not due to "dysarthria," but are produced by defective conceptions of the structure and rhythmic balance of the symbol, which interferes not only with articulated speech but also with internal verbalization. All these functions are not uniformly disturbed in every instance, and aphasics differ profoundly in their clinical manifestations. The majority, especially in the earlier stages, show evidence of widespread defects in the use of language; the more acute and severe the lesion, the graver and more extensive will be the disorder it produces. Many of these changes may disappear, and sometimes the patient recovers entirely; but, in serious cases, one or more aspects of symbolic thinking and expression remain affected over a considerable period, and the aphasic manifestations consequently assume some particular form.

Analysis of these clinical varieties seems to show that the various functions included under the term "symbolic thinking and expression" can be dissociated in different ways under the influence of organic injury. They do not correspond to different stages of dissolution or recovery; nor do they reveal directly the elements out of which language is built up. On the contrary, they show the components into which a highly complex set of psychical processes can be separated by destruction of certain portions of the brain. Provided we bear these principles in mind, we are justified in grouping the clinical manifestations under separate headings. Each case of aphasia is the result of the loss of one or more of these groups of functions; fortunately, all of them are rarely affected in the same patient, for, if this were the case, he would be reduced to a state in which all detailed examination would be impossible. Thus, in each patient we must determine which functions are disturbed and which have remained unaffected. By careful analysis it seems possible to separate these disorders of speech into four main varieties; but it must be remembered that they can appear together in every combination.

To each of the groups I have given the name chosen from the most salient defect in the use of words.

(1) *Verbal aphasia.*—This is essentially a disturbance of word formation; words are evoked with difficulty, the patient's vocabulary is greatly restricted, and enunciation is slow and halting. Writing tends to show the same sort of errors as articulatory speech and spelling is defective. The patient has difficulty in reading to himself with pleasure, because he is unable to retain in his memory a long series of words.

Numerals are affected to a slighter degree; their significance may be recognized and acted on correctly, although they are wrongly enunciated. As speech returns, commands given in spoken or printed words can be executed, but orders which necessitate the evocation of some word or phrase may be carried out badly. These patients recognize, however, whether the task they are attempting has been performed correctly or not. They can draw, play card games, and appreciate jokes set out in print or in pictures. It is this group of defects which is mainly responsible for the condition described by Broca as "aphemia" and usually spoken of as slight "motor aphasia."

(2) *Syntactical aphasia.*—This is an easy form to distinguish because the patient tends to talk jargon; not only is articulation of the word ill-balanced but the rhythm of the phrase is defective, and there is want of grammatical coherence. Speech, once started, is voluble and words are emitted with great rapidity; sometimes each one is comprehensible, however difficult it may be to gather the full meaning of the phrase; but in other cases the words uttered are pure jargon. The power of naming objects placed before him may be retained by the patient, in spite of the jargon by which he is hampered. Not infrequently, when he cannot utter some word, or when the sound emitted is incomprehensible to his auditor, he can write the name correctly. These patients can read if they are not compelled to reproduce the meaning in words; writing is usually less affected than external speech, and single words, especially names in common use, can be written correctly; but any attempt to convey a formulated statement is liable to end in confusion. This form was described by Hughlings Jackson, but has not received the attention it deserves except in the writings of Pick.

(3) *Nominal aphasia.*—This is essentially a defective use of names and want of comprehension of the nominal meaning of words or other symbols. The patient reads with extreme difficulty, especially if he attempts to spell out the words; writing is gravely affected, but he may be able to copy print into cursive handwriting. Writing to dictation and all actions demanding choice are performed with difficulty to spoken command; counting is possible to a varying extent, but the significance of numbers, the power to carry out simple arithmetical operations, and appreciation of the relative value of money are usually more or less affected. The patient is unable in most cases to draw an accurate ground plan of some room with which he is familiar; he cannot play cards, but chess and draughts may be possible. This form cannot be fitted into any of the older methods of classification; for, on the one hand, it has

some of the characters of "motor aphasia," whilst, on the other, other defects would have been attributed to "sensory aphasia." The separation of word-formation from naming and its allied functions is an entirely new feature in the classification of the aphasias.

(4) *Semantic aphasia.*—So far, the names I have applied to the various forms of disorders of speech have borne some relation to the verbal defects; for this group, however, it is difficult to find a suitable designation that will express the essential nature of the disturbance which extends beyond the limits of organized words. I have chosen the term "semantic" as a label for this form of aphasia, because the affection comprises want of recognition of the full significance or intention of words and phrases. The patient may understand each word or short phrase exactly as he can comprehend the details of a picture, but the ultimate meaning escapes him. He fails to grasp the final aim or goal of the action imposed upon him from without; he cannot formulate symbolically a general conception, although he can enumerate the details of which it is composed. He can read and write, but the result tends to be inaccurate and confused; counting is possible and the value of numerals can be recognized, but arithmetical operations are impossible, or are carried out uncertainly and with difficulty. The patient is unable to add or subtract, because the mathematical process itself is incomprehensible. He fails entirely to comprehend most jokes, especially if they demand the complete understanding of a picture or its legend. He cannot play card games or put together puzzles, which confuse him greatly. These semantic disorders interfere seriously with the actions of daily life and render the patient useless for any but the simplest employment; and yet his memory and intelligence may remain on a comparatively high general level. He does not forget people or places, and can recall accurately events, both recent and remote; but he has lost the power of appreciating the ultimate or non-verbal meaning of words and phrases, and fails to recognize the intention or goal of actions imposed upon him.

I have attempted to put before you shortly a conception of the phenomena of aphasia, based on a systematic re-examination of the clinical facts by new methods. The results I have obtained are of no direct practical value to the physician, but they form a fascinating example of the interaction of body and mind—one, moreover, capable of experimental demonstration. This problem has occupied the attention of the acutest human intellects for over two thousand years. The schoolmen answered the eternal riddle in material terms founded on

imaginary anatomy. They were followed by the philosophers, who, for the most part, spun their theories out of *a priori* conceptions. Then came the era of the physicians, who were unwillingly forced to face the question, because the most illuminating examples of the effect of bodily conditions on the state of the mind were entrusted to them for therapeutic supervision. They developed an extensive and accurate acquaintance with the anatomy of the brain, but employed the fruits of this great advance in knowledge in the service of an obsolete and almost mediæval psychology. Fifty years ago Hughlings Jackson raised the question of disorders of speech to a higher plane by rejecting all *a priori* conceptions; but he stood alone and medicine turned a deaf ear to his teaching. To-day, however, the older psychology is tottering to its grave, and I am proud to think that a long continued and arduous series of researches has led me at last to understand that great empiric philosopher, the founder of English neurology.

REFERENCES.

[1] BAGINSKY. "Aphasie in Folge schwerer Nierenerkrankungen," *Berl. klin. Wochenschr.*, 1871, S. 441.

[2] BASTIAN, H. CHARLTON. "On the Various Forms of Loss of Speech in Cerebral Disease," *Brit. and Foreign Med.-Chir. Rev.*, 1869, January-April, xliii, pp. 209-236, 470-492.

[3] *Ibid.* "On some Problems in connection with Aphasia and other Speech Defects," (the Lettsomian Lectures), *Lancet*, 1897, i, pp. 933, 1005, 1131, 1187.

[4] *Ibid.* "Aphasia and other Speech Defects," Lond., 1898.

[5] BROCA, P. "Perte de la parole. Ramollissement chronique et destruction partielle du lobe antérieur gauche du cerveau," *Bull. Soc. Anthropol.*, 1861, 18 avril, ii, pp. 235-238.

[6] *Ibid.* "Remarques sur la siège de la faculté du langage articulé, suivies d'une observation d'Aphémie," *Bull. Soc. Anat.*, 1861, vi, pp. 330-357.

[7] *Ibid.* "Nouvelle observation d'aphémie produite par une lésion de la troisième circonvolution frontale," *Bull. Soc. Anat.*, 1861, vi, pp. 398-407.

[8] *Ibid.* "Sur la siège de la faculté du langage articulé," *Trib. Méd.*, 1868, No. 74, pp. 254-256, and No. 75, pp. 263-269.

[9] "Discussion sur l'Aphasie." *Rev. Neurol.*, 1908, xvi: Séance du 11 juin, p. 611; Séance du 9 juillet, p. 974; Séance du 23 juillet, p. 1025.

[10] GALL, F. J. "Craniologie ou découvertes nouvelles du Docteur F. J. Gall." Ouvrage traduit de l'Allemand, Paris, 1807.

[11] GALL, F. J., and SPURZHEIM, G. "Recherches sur le Système Nerveux en général et sur celui du Cerveau en particulier," Paris, 1809.

[12] *Ibid.* "Anatomie et Physiologie du Système Nerveux en général et du Cerveau en particulier, avec des observations sur la possibilité de reconnoître plusieurs dispositions intellectuelles et morales de l'homme et des animaux par la Configuration de leur Têtes" (4 vols. in folio, 4 vols. in quarto, atlas of 100 plates), Paris, 1810-19.

[13] HEAD, HENRY. "Aphasia and Kindred Disorders of Speech," *Brain*, 1920, xliii pp. 91-107.

[14] JACKSON, J. HUGHLINGS. "Papers on Aphasia and Kindred Affections of Speech," reprinted in *Brain*, 1915, xxxviii, pp. 1-190.

[15] LICHTHEIM, L. "On Aphasia," *Brain*, 1885, vii, p. 433.

[16] MARIE, PIERRE. "Revision de la Question de l'Aphasie," *Semaine méd.*, 1906. (1) "La 3e circonvolution frontale gauche ne joue aucun rôle spécial dans la fonction du langage," 23 mai, pp. 241-247; (2) "Que faut-il penser des aphasies sous-corticales?" 17 octobre, pp. 493-500; (3) "L'aphasie de 1861 à 1866; essai de critique historique sur la genèse de la doctrine de Broca," 28 novembre, pp. 565-571.

[17] MOUTIER, F. "L'aphasie de Broca" (Travail du laboratoire de M. le Professeur Pierre Marie), Paris, 1908.

[18] PICK, ARNOLD. "Die agrammatischen Sprachstörungen: Studien zur psychologischen Grundlegung der Aphasielehre," 1 Theil (the second part has not appeared), Berl., 1913.

[19] SUDHOFF, W. "Zur Lehre v. d. Hirnventrikeln in textlicher v. graphischer Tradition d. Altertums u. Mittelalters," *Arch. f. ges. d. Med.*, 1913, vii, S. 149.

[20] VILLERS, C. "Lettre de Charles Villers à Georges Cuvier sur une nouvelle Théorie du Cerveau par le Docteur Gall; ce viscère étant considéré comme l'organe immédiat des facultés morales," Metz, an x (1802).

[21] WERNICKE, C. "Der aphasische Symptomencomplex," Breslau, 1874.

[22] *Ibid.* "Ein Fall von isolierter Agraphie," *Monatschr. f. Psych. u. Neur.*, 1903, xiii, S. 241.

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President—Dr. ERNEST S. REYNOLDS.

Meeting held November 11, 1920.

Discussion on Aphasia.

DR. HENRY HEAD, F.R.S., said that (1) it was universally acknowledged that speech could be affected by destruction of the substance of the brain. If the lesion was acute, all aspects of the act of speech might be generally reduced. As the nervous tissue recovered, all these functions might return more or less together, and the aphasia disappeared without the manifestations assuming at any time a specific form. This generalized loss of function was particularly liable to occur when the disorder of speech was produced by vascular lesions or by any condition that lowered the vital activity of the brain. In cases of head wounds it was therefore important to wait until the original nerve shock had passed away and the wound had healed, before attempting to draw any conclusions as to the supposed specific character of the loss of speech. It might happen, however, after the acute stage has passed away, that the lesion of the brain was found to have affected the production of those physiological activities which underlay one or more special aspects of the perfect use of language and allied functions. Speech consequently assumed in such cases a clinically specific form.

(2) The categories of such partial forms of aphasia could only be drawn up in terms of the tests employed; they did not correspond to exclusive loss of speaking, reading or writing, nor could they be divided into "motor" or "sensory" abnormalities. As the result of serial tests, he had attempted to show that no descriptive term can be found which covered all the functions disturbed in one or other forms of aphasia and kindred disorders of speech. For amongst these functions were many which were not usually associated with speech. He had

therefore adopted the name "Symbolic Thinking and Expression," because it represented that group of psychical activities which was most severely affected by unilateral lesions of the brain. Analysis of the clinical varieties of aphasia seemed to show that the various functions included under this term could be dissociated in different ways under the influence of organic injury. They did not correspond to different stages of dissolution or recovery: nor did they reveal directly the elements out of which language was built up. On the contrary they showed the components into which a highly complex set of psychical processes could be separated by destruction of certain portions of the brain. Something was lost which made perfect speech impossible, and the whole act assumed an abnormal form. Part of the manifestations were due to the negative aspect of the disturbance, part to the disorderly activity of function which remained.

(3) Before attempting to determine what portion of the brain when injured was associated with the different clinical affections of speech, it was necessary to discover by serial observations the nature of the psychical disturbance in each particular case. In the past attempts had been made to localize the anatomical site of the lesions responsible for "motor" or "sensory aphasia, "alexia" or "agraphia." But these *a priori* terms did not correspond to any specific functional categories discoverable in cases of these disorders of language due to a unilateral lesion of the brain. The fact that specific clinical forms of aphasia occurred showed that some lesions affected one aspect of the language mechanism more than another. He was prepared to enter on this question later, but was anxious that the present debate should be concerned with the nature of the loss of function produced by a unilateral lesion of the brain and with the clinical forms it might assume.

Dr. JAMES COLLIER: In opening this discussion upon the subject of "Aphasia," I must in the first place thank Dr. Head for the opportunity which he has afforded us and for his kindness in asking me to address you first. There has been for many years a growing revolution against the former conceptions of the nature of the speech function and the explanation of its defects, which has led to much bitter controversy, and has culminated in Dr. Head's bold endeavour to place this subject upon a sounder and truer foundation. It is very important in my opinion that we should have this opportunity of discussing this subject with him, and in this discussion there is no obligation that we should be restricted to the subject matter of his recent publications.

The necessity for this revolution was great. The literature is enormous and bewildering, and the types of aphasia multitudinous. Take for example Lichtheim's seven types based upon a conception of centres of localized function connected by isolated paths which only exist in theory, and with which types our clinical cases never truly correspond. Dr. Head would make a clean sweep of former conceptions and types of aphasia and introduce a newer conception of the functions of the cortex of the brain in speech, a more refined method of observation and a new classification. For the greater part I am in agreement with him. In some details I differ from him, while in others I am not in the position to express an opinion.

We have to bear in mind in dealing with the function of speech and its defects that we are considering the most highly evolved and complicated function of the brain, closely bound up with intellect itself, the mode of working of which is probably beyond any flights of human conception. And any analysis which we can make into this function must be of necessity crude and liable to misunderstanding. Conceptions of the coarse localization of function in the physiological substratum must, it seems to me, be erroneous, and any attempts to formulate the working of this intricate mechanism in diagram are fundamentally absurd.

I shall direct your discussion therefore to :—

(1) Your views upon the matter of localization.

(2) Your opinions as to the utility of retaining any of the time-honoured terminology.

(3) Would you retain any of the types of aphasia as true clinical types ?

(4) Your views upon Dr. Head's method of examination and upon his classification.

The former conceptions of aphasia were based upon the most able clinical and pathological investigation of great men. They have served to teach us, and, so far as the localization of gross cerebral disease is concerned, they have led us to almost as great an accuracy in the regional diagnosis of local disease as we are ever likely to reach.

It is interesting to consider for the moment what were the grounds upon which the conceptions of isolated centres concerned with the registration of visual word images, auditory word images, and executive speech function were built up. It was from the study of comparatively large anatomical lesions, always subcortical as well as cortical, and generally more subcortical than cortical, and when such lesions were

situated in such a position upon the confines of the cortical speech region as to sever more or less completely the usual paths of inlet to the speech region, either upon the visual side or upon the auditory side, or blocked the paths of exit upon the executive side, the most obvious of the speech defects resulting were failure of acceptance of speech on the visual side, word-blindness, or on the auditory side, word-deafness or failure of exteriorization of speech—motor aphasia and agraphia.

It followed that these limited regions—the angular gyrus, the mid-temporal region, and the third and second frontal convolutions—were labelled as visual word-centre, auditory word centre, and motor speech centre. I submit therefore for your discussion that when the conditions which have been termed "word-blindness," "word-deafness," "motor-aphasia," and "agraphia" occur, any one of them, as the most obvious result of a lesion of the brain, that they are the result of isolation of the speech region of the cortex upon the incoming sides and upon the outgoing sides respectively, and are not due to the destruction of local memory centres. It follows from the nature of the lesions that when these incoming and outgoing paths are damaged, there is often considerable damage to the cortex itself with the appearance of speech defects of a different order. Dr. Head would rid us of these terms, "word-blindness," "word-deafness," and "motor aphasia." Yet they are the terms which have been long in use, and while freely admitting that they may be fundamentally incorrect, physiologically inadequate and usually untrue clinically, yet these terms do roughly describe the most obvious features of many speech defects. They have proved useful for teaching purposes and will prove hard to replace.

In the Hughlings Jackson lecture Dr. Head doubts whether there are any such conditions as "pure alexia," "pure aphasia," and "pure agraphia," and in this attitude I am unable to follow him. For whatever conception we may have of the localization of function in the cortical speech region, we are bound to admit the existence of afferent paths to the speech region upon the auditory side and upon the visual side, and efferent paths therefrom by which speech is usually exteriorized, both spoken and written, and in these paths there is surely strict anatomical and physiological separation. In so far as these paths exist as definite anatomical tracts for a sufficient distance to allow of the isolated involvement of any one of them by a lesion, we cannot deny the possibility of such a lesion, nor that the resulting speech defect is more or less narrowly confined to one aspect of speech.

I hold that the clinical condition described by Dejerine as "pure alexia" or "pure word-blindness" and attributed to a lesion at the posterior limit of the speech area, which severs the connection of that area from the visual area, is one of the most certain and definite of clinical conditions. I have seen it many times and have seen the theoretical lesion verified on more than one occasion.

Similarly I see no reason why the conditions of pure agraphia and pure aphasia also described by Dejerine should not be true clinical conditions. Their acceptance does not affect our conceptions of the cortical functions of speech. There must be both physiological and anatomical separation on the way out of the paths by which spoken and written speech is exteriorized, and the possibility of isolated damage to such paths must be admitted to be possible. I have seen one case which during the early moments of his seizure was certainly a case of "pure agraphia." This was a man who was lying in the National Hospital with a fracture-dislocation of the back, who in the course of writing a letter to his wife was seized with a disability to write, to which he immediately had my attention called, and for the space of more than an hour, during which time his condition was investigated by myself and by Dr. Rennie, now of Sydney, he presented a defect which exactly corresponded to "pure agraphia." After which his lesion extended widely, and proved rapidly fatal. We found in this case a subcortical lesion beneath the lower frontal convolutions associated with an extensive thrombosis of the Sylvian artery, and I think it is not too much to claim that the commencement of the subcortical lesion was in such a position as to block the outgoing path for written speech, and did actually produce a condition of "pure agraphia." I agree that pure "word-deafness" does not exist as a clinical entity, and I would explain this fact in that it is unlikely for any lesion to sever the auditory path entering the speech region of the cortex without interfering grossly with that cortex both anatomically and physiologically. Yet there are cases which all of us must have seen in which lack of acceptance of spoken speech stands out in undue contrast with the acceptance of visual speech, and I would attribute this isolation of function not to any separate location of function in the speech cortex, but to blocking of the auditory afferents.

I presume that the chief paths entering the cortical speech region do not end abruptly, nor do the outgoing paths begin abruptly, but rather gather in their course, and that therefore the element of blocking must be an important factor in the production of the speech defects under consideration.

There is another consideration in connection with the afferent paths which I deem of great importance, especially in connection with those speech defects which Dr. Head terms "syntactical." When we speak aloud we have an immediate recognition of what we say by means of the auditory path, which allows of analysis of the correctness and fitness of what we have said and of its correction if faulty. This backlash, if I may be allowed the expression, is of great importance during the learning of speech in any form, and it surely must be a great factor when we speak, in keeping the physiological activity of speech and thought at a high level.

There is also another backlash in the way of the sensations accompanying the movements of the executive mechanism, but this plays but a very minor part in the speech of normal persons, though since in the deaf-mute who has learned to speak by lip reading it is his only guidance both in the learning of his speech and in its execution, this factor must not be forgotten.

There is of course a similar constant visual appreciation and correction in writing.

Now, when on account of the lack of acceptance of spoken speech this immediate consciousness of the correct execution of speech fails, surely this must throw the mechanism of thought and speech into dire confusion. I submit that this is the essential cause of those defects of speech which Dr. Head groups as "syntactical," and certainly of such conditions as voluble jargon-aphasia. I shall refer to this subject again later on.

Dr. Head in his Linacre Lecture seems to favour some vague separation of function, some very general localization of functions in the cortical speech area which he does further comment upon. If by this he means a gradual passing over from behind forwards from receptive functions to executive functions, I am inclined to agree with him. I take it that he would expect "verbal defects" from lesions farther forward than those productive of "nominal" and "semantic" defects. If on the other hand he means any separation of localization for visual and auditory speech functions, then I disagree.

It seems to me that the function of visual speech, acquired at a much later age than is auditory speech, tacked on as it were to an already perfect and deeply impressed function of auditory speech, learnt by means of an already well versed auditory speech function, must occupy the same physiological substratum as does the auditory speech function, and is separately localized only in so far as its chief afferent and executive paths are concerned.

I submit for your discussion, that, as the result of lesions of the cortical speech region in any of its parts, there is likely to be depression of its physiological activity as a whole, and that the expression of this depression may be a major impairment or loss of its less deeply impressed functions. For the speech functions are not equally impressed nor equally stable. The auditory speech functions both upon the receptive and upon the expressive side are in most people much more deeply impressed and more stable than are the corresponding visual speech functions. And I submit that this accounts for the impairment of loss of visual speech recognition which almost always occurs in lesions of the anterior regions of the speech cortex, and that it accounts also for the major loss of the visual speech functions which is the rule in lesions situated farther back.

I have in two cases observed the practically complete recovery from aphasia in polyglots who were afflicted with syphilitic vascular disease of the speech region, and in each case recovery was much more rapid in the more deeply impressed native tongue, and less rapid and less perfect in the language more recently acquired, and less long used. And when speech was practically recovered so far as the native tongue was concerned they still presented gross defects in the foreign tongue. It would be fair to say that at a certain stage of recovery they were aphasic in one language and not in another, and this must surely be explained by the dropping out of the less impressed function when the activity of the speech functions is lowered, as the result of disease.

Dr. Head calls particular attention to the highly important and certainly correct principle that the alterations in speech function which result from lesions of the brain are not in terms of the primary elements out of which speech has been evolved, nor do they reveal the elementary basis of the acts of speaking, reading and writing, but that they are in terms of the complete and highly developed function, which fails progressively from its highest to its lowest with increasing degrees of damage.

Though in infancy we learn our speech in terms of words and short phrases, and our writing in terms of letters and words, yet when speech is highly developed we speak and receive speech in a running pattern of sounds of which the individual elements are disregarded. We do not in reality speak, hear, read or write in words, but in a running pattern comparable and often inseparable from the running patterns of thought. It is the pattern which is exteriorized and accepted as a whole. The individual words of which this pattern is made up

singly convey little or nothing of meaning unless we put them into another pattern by parsing them. The running patterns of speech are of great importance in the consideration of speech defects. In their course they reinforce and raise the level of activity of speech and thought, and they have an important momentum. The common example of the aphasic who is unable to use a particular word on command, and yet produces it without effort in the run of the pattern by which he is expressing his inability, or trying to talk round the subject, well illustrates this point. In setting a speech pattern going he gets a run at his difficulty just as in taking a running jump one gathers the necessary momentum to overcome the difficulty. Deformity and distortion of these patterns are common phenomena among speech defects, and the difficulty to set them in orderly running is fundamental.

Sometimes one is surprised in examining a case in which speech acceptance is seriously impaired, to find that the patient will gather considerable meaning from a phrase when he does not do so from simpler forms of approach. He accepts something from the pattern when simpler forms of communication do not appeal to him.

Dr. Head has laid before us in the Linacre Lecture a scheme for the examination of patients suffering from defects of speech which seems to me in every way admirable and worthy of universal adoption. It provides for the examination of the speech functions in the varying states of their incapacity from the lowest to the highest. The serial tests are calculated to rouse into activity depressed functions which otherwise give no sign of their existence, as for example the patient who though unacceptive to visual speech in any ordinary way matched a colour with its printed name, thereby showing that some dim function of visual speech was rousable by the combined presentation of the colour and its name. Of especial interest as arising from the serial tests is the proposition that inasmuch as an act requires verbalization for the clear recognition of its nature, the act tends to fail in execution if verbalization is defective. This proposition emphasizes the importance of verbalization in the concept. It is so far as I am aware a new proposition—at any rate it is new to me and seems to me to be of far-reaching importance in the interpretation of speech defects.

The serial tests are excellent, the difficulty comes with the interpretation of the results. The classification of speech defects into "verbal" defects, "nominal" defects, "syntactical" defects and "semantic" defects requires careful consideration. Dr. Head's position is clearly

defined. He starts with the conception of a region of the cerebral cortex concerned with speech functions which habitually acts as a whole and not in terms of isolated localized functions. This position seems to me correct and unassailable. He makes an analysis from the clinical side only and commits himself to no opinions of anatomy and localization, but deals entirely with objective physiological defects in his attempt to elucidate the manner in which the speech function breaks up as the result of lesions of the brain. Whatever views we may hold individually upon the nature of the speech functions, such an analysis is not controversial. It can offend no one. It certainly is very valuable. It rests upon a sound scientific basis. In making this analysis he used specially picked cases. Selected firstly because of slight and partial lesions which leave a great part of the anatomical substratum of speech in a relatively physiological condition, and therefore are much more suited for analysis in that they yield much more information. Selected secondly because from the nature of the lesion the cortex of the brain may be deemed to have been particularly affected, though this selection may give rise to doubt as to whether the lesion in any given case was mainly cortical; it is open perhaps to less objection in this respect than are the vascular and other gross lesions in civilian cases upon which great theories have been built up. And lastly selected because they were suitable for the analysis—all cases are excluded in which images are lost. Such an arbitrary selection must be allowed if we are to get a clear issue and avoid confusion. The results of the analysis are recorded in minute detail. There is no attempt at explanation, and no theory is raised upon them. Localization is not considered as this is an anatomical matter. The results are in the present state of our knowledge useful in enumeration only. The classification is a list of symptoms which from their nature tend to fall into four groups, and the patients tend to fall into four corresponding groups.

Dr. Head submits that these groups are the expression of the manner in which the speech function breaks up as the result of the slighter degrees of damage to the cortical substratum subserving that function. Dr. Head's position does not admit of much contention or controversy. It does not in any way subvert or undermine the correctness of the rough methods by which all of us are accustomed to localize gross cerebral disease with accuracy when the speech region of the cortex is involved. It strikes fundamentally at former theories both of the nature of the speech function, and at the localization of separated functions—not many of which I submit have been truly applicable to clinical conditions nor theoretically satisfactory.

Comments upon his division of symptoms and his classification of cases into the four groups cannot be adequately made until we have applied the serial tests to our suitable cases and made our own deductions therefrom in the light of this classification. Whether this grouping does really represent the manner in which the cortical speech function may disintegrate is to me uncertain, for by no method of introspective analysis, no consideration of the development of speech functions, and by no anatomical or physiological considerations can I see why it should do so.

I shall conclude these remarks in referring to two points in connection with his classification which I deem of considerable importance. In connection with verbal aphasia it is quite refreshing to see it laid down with emphasis that deformity of words in the way of mispronunciation, omission and misplacement of syllables—abnormal word formation, in fact—is characteristic of this condition, and further that the same deformity that occurs in the spoken words is met with also in the written words, proving the inseparability of these two functions. This is a clinical fact which has been neglected or passed over by so many writers upon aphasia. Sometimes this deformity is so great as to make the speech almost irrecognizable and in a way a jargon-aphasia. I have been in the habit in the past of referring to this condition as "anterior" jargon-aphasia from the forward situation of the lesion which is usually responsible for its production. It is of course instantly distinguishable from true jargon-aphasia. I am not sure that the essential rhythm of word and of phrase in this condition is always intact as Dr. Head thinks. I believe that considerable defect of rhythm may occur and that the phrase may be deformed as a whole just as the word is deformed.

In syntactical aphasia as a result of damage to the speech region of the cortex, I find myself for the first time in the course of these remarks quite opposed to Dr. Head; it is his meaning that such a condition can arise from a lesion of the cortex. True jargon-aphasia is a thing quite of itself. It is the only condition among speech defects in which overaction is manifest. The volubility which is characteristic of this condition is extraordinary and seems to increase the more severe the jargoning of the speech becomes. Even from the first there may not be that general depression and difficulty of arousing the speech functions that is seen in connection with other forms of speech defect. The intelligence and quickness of apprehension of all uncomplicated cases of jargon-aphasia even in its severest degrees stand out. The condition seems to

me that of a function severed from control and without guidance. I must admit that I have not had the opportunity of examining cases with such mild degrees of this condition as has Dr. Head. I have never in any case of this nature seen so great a degree of speech acceptance upon the auditory side as some of his cases had. In all my cases there has been severe blocking upon the auditory side.

I may here refer to two cases almost identical in nature which were under my observation and which were of so striking a nature as perhaps to have biased me unduly in my conception as to the nature of jargon-aphasia. These were both men who at their work were seized with a slowly oncoming jargon-aphasia which in the course of a few hours rendered speech incomprehensible. There was no ictus with the onset, no affection of intelligence, and these men did not even stop their work. They were brought to me within a few days—the one by the police for making a disturbance in the street, the other by his wife, as being mad. They were both uncomplicated cases of severe jargon-aphasia. The one was absolutely non-acceptive of speech either upon the auditory or upon the visual side, and the other was non-acceptive upon the auditory side, but appreciated visual speech to a considerable extent. One of these patients presented optic neuritis, and the other soon developed it. Both of the cases showed on pathological examination a well-defined tumour undercutting the posterior and mesial aspects of the temporal lobe, comparatively far from the cortex which was nowhere involved. The tumours were in such a position as might reasonably be expected to sever the auditory afferent path to the speech region in both cases, and in the first case the tumour curved round towards the cortex in the angular region, and is likely to have severed the visual afferents.

The striking points about these two cases were :—

The onset of jargon-aphasia as the initial symptom.

Its rapid development to an extreme degree.

The lack of acceptance of auditory speech.

The peculiar subcortical position and discrete nature of the lesions which nowhere involved the cortex.

It was difficult to regard these cases in any other light than that of isolation phenomena in which the auditory balance and rhythm which is so essential a part in the production of speech was lost owing to the interruption of afferent paths to the speech region of the cortex, by which speech exteriorization is habitually guided. I submit that it is the lack of the immediate impressions of correct execution which we

receive when we speak, both with our ears and from the muscular movements of speech, which is responsible for the condition of jargon-aphasia. It is the expression of a function which has lost control and which has "gone mad" from isolation.

Surely these cases of jargon-aphasia are primarily unaware of their defect of utterance by reason of lack of auditory acceptance, some of them partially perhaps, but the majority of them deeply. They do not show the embarrassment and loss of confidence that such defects, if recognized, would certainly cause, though they may show some embarrassment and emotion from lack of appreciation of what they are trying to say in their audience. It is true that in one of Dr. Head's cases there was a greater degree of auditory acceptance than I have ever seen in a case of jargon-aphasia, but I gather that all of them were more or less defective in this way, but to what degree he does not make very clear.

Visual speech and writing are almost always affected to a less degree than auditory speech in cases of jargon-aphasia, and this I attribute to preservation of the visual afferents. One of Dr. Head's cases seems to show that correct writing can occur without verbalization—I refer to the case who on being shown a matchbox uttered a three-syllable jargon and promptly wrote the word matches correctly. This is a very interesting and unusual fact, for writing is usually strictly dependent upon verbalization, and when verbalization is imperfect the writing shows exactly the same defects as does speech. I agree with Dr. Head that verbalization is always very imperfect, and in the more severe degrees of the condition writing is almost as much affected. Now, this relative escape of the visual word function is surely the reverse of what occurs in cortical lesions, in which, I believe, the visual word function always shows a major defect compared with that of the auditory word function, and I use this as an argument against the cortical origin of jargon-aphasia.

Further, jargon-aphasia is a comparatively rare condition. It is hardly ever met with in the common lesions of the temporal region from vascular disease of the Sylvian artery where the temporal cortex is likely to be involved. I think also that when jargon-aphasia is due to a progressive lesion, an extension of the lesion to the temporal cortex causes the jargon-aphasia to lessen in volubility and finally to disappear.

I submit that an intact cortex is necessary for the appearance of this form of speech defect, and that it is a pure type of aphasia, and like the other pure types of aphasia is referable to an isolation from auditory afferents and to a subcortical lesion.

Sir James Purves Stewart: We are so accustomed to receive original and stimulating thoughts from Dr. Head that whenever he brings us something fresh many of us tend to be prejudiced in advance in his favour. This, of course, being a mere feeling-tone, partly thalamic, and not a process of cortical reasoning, is just as wrong as it would be if we were to start prejudiced against him.

At the outset let me say that Dr. Head's conception of aphasia is full of instruction to us all. No one will venture to cast any doubts on Dr. Head's clinical observations. Every one of us must be grateful for the new scheme of investigation which he has presented to us and for the various interesting clinical facts which this scheme has brought out. We have studied with interest his proposed new classification of aphasia into four varieties, viz. :—

(1) Verbal aphasia. Defective word-formation, both in internal and external speech.

(2) Nominal aphasia. Defective use of nouns, both spoken and written.

(3) Syntactical aphasia, with want of coherence and a tendency to talk jargon.

(4) Semantic aphasia. Deficient comprehension of the full significance of words and phrases. Although the patient can speak and write, he cannot formulate a general conception but only the details of which it is composed.

This new classification is of the highest psychological value. But there seems to me no adequate reason why we should necessarily throw overboard all our previous clinical experiences, or our division of aphasia into two great classes, viz. :—

(1) *Psycho-motor aphasia*, aphasia of expression, or verbal apraxia.

(2) *Psycho-sensory aphasia*, aphasia of comprehension, or verbal agnosia.

More especially as these two great clinical divisions correspond to well-established anatomical facts.

One thing we note in Dr. Head's recent paper is that he abstains, so far, from giving us particulars of the anatomical site of the brain-lesion in a single one of his cases.

May I be allowed, as an ordinary clinician, to relate a few clinical examples of aphasia. All except one have been selected, like Dr. Head's cases, from young men suffering from war-lesions of the brain.

Case 1.—W. M., aged 55. Lower frontal lesion.

This patient was admitted to the Westminster Hospital with signs of acute cardiac failure due to old aortic disease. He had no paralysis of the face or

limbs and was able to speak fluently. His pulse was almost imperceptible and the heart sounds were almost inaudible, forty-eight per minute.

Twenty minutes after his admission to the ward, he suddenly became speechless and his face became asymmetrical. His intelligence was unimpaired. He was able to understand and execute all sorts of verbal commands, to put out his tongue, to place his hand on any part of his face or head, to move the right or left thumb by bending it half-way or straightening it out, as requested. But he was unable to utter any word whatever. The right face was weaker than the left. There was no weakness of any limb, but the right plantar reflex was extensor in type and the right abdominal reflex was absent.

Two days after admission he died, with symptoms of increasing cardiac weakness, cyanosis and rhythmic breathing of stertorous type.

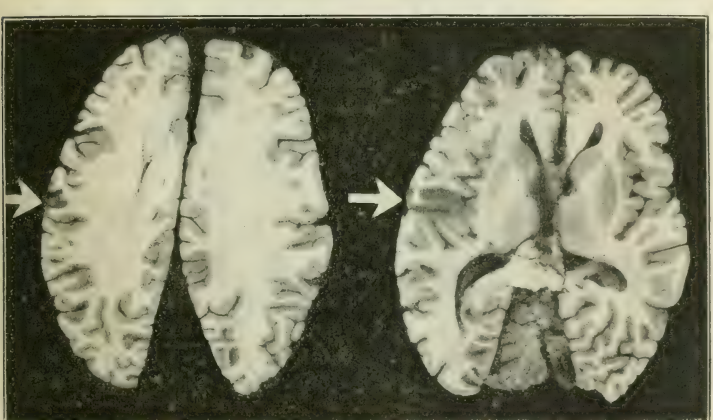

FIG. 1.—Left lower pre-central lesion.

At the autopsy, there was an old syphilitic aortitis, and just above the aortic valves was a large tag of thrombus about an inch long and one-eighth inch thick.

The brain, after hardening, showed a small area in the lower third of the left precentral gyrus, in which there was a dark marbled appearance, due to embolic blocking of a small cortical arteriole (see fig. 1). This lesion extended deeply into the operculum, but did not affect the insula, nor the basal ganglia, i.e., Marie's "lenticular zone" was unaffected. There was no other lesion elsewhere in the brain.

This is an uncomplicated case of psycho-motor aphasia. Probably Dr. Head would classify it as a verbal aphasia.

Case 2.—W. T., lieutenant, aged 24. Mid-Rolandic lesion, pre- and post-central.

The patient was wounded by a ricochet bullet in the left parietal region. He was knocked down, but not unconscious. His speech at once became jumbled. After a quarter of an hour he noticed paralysis of the right arm, and after about four hours the right leg also became paralysed.

A week later he was trephined, and two pieces of bullet were removed from the surface of the dura. The dura was not opened.

The patient states that after his injury he could not get out his words at all, nor could he understand things properly.

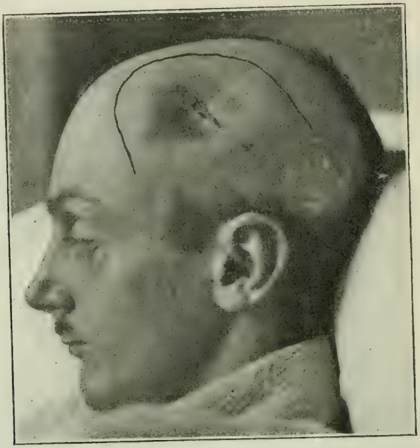

Fig. 2.—Left pre- and post-central wound.

On examination, five weeks after the original injury, he was highly intelligent and had no evidence of word-deafness or word-blindness. He understood and executed accurately verbal requests and also written requests, e.g., "Close your eyes and then put out your tongue."

He was able to write spontaneously and to dictation short sentences with his left hand, copying accurately, but occasionally making mistakes in spelling to dictation, e.g., "hopsital" for "hospital."

He recognized all objects shown to him and named them correctly, but tended to miss out the initial consonant, e.g., "'overeign, 'illing, 'atch-box." But he repeated words and phrases correctly to dictation. He added up columns of figures fairly accurately and read the newspapers intelligently.

The right face was slightly weaker than the left. There was severe paralysis and spasticity of the right upper limb and slight weakness of the

right foot. There was astereognosis of the right hand and foot. With the right hand he recognized an object like a coin as hard and round, but could not name it. The deep reflexes were increased in the right upper and lower limbs, with right-sided ankle-clonus. The right plantar reflex was extensor in type and the right abdominal reflex absent.

A month later he had improved a great deal. He now only hesitated very slightly, chiefly with initial sounds. Sometimes he misplaced syllables, but chiefly when excited. He felt himself mentally brighter, but thought he was a little slower than before his injury, e.g., when counting money.

He understood everything said to him, and executed accurately written commands. He copied accurately from printed to written letters and wrote to dictation (left hand) without making spelling mistakes. He added up figures, both simple figures and £ s. d., e.g.,

£2	17	9
8	13	4
£11	11	1

He repeated words dictated to him, but sometimes mispronounced syllables, e.g., said "February twenty-fith." He named objects shown to him accurately, also read aloud accurately from a book.

The weakness and astereognosis of the right upper limb were still severe, but he was now able to walk, dragging the right leg.

We clinicians would call this an example of psycho-motor aphasia. Dr. Head would probably classify it as a verbal aphasia.

Case 3.—D. M., aged 31. Inferior frontal lesion.

This patient was seen at Salonika. Eight days before I saw him he was admitted to hospital unable to speak, but able to understand what was said to him and able to write with his right hand, jumbling his words in writing.

He had a depressed fracture of the skull in the left upper temporal region (see fig. 3) over the region of the lower end of the Sylvian fissure, apparently from a glancing shrapnel wound. There was a large lacerated scalp wound, and the fracture was found to have fissures radiating backwards for a short distance. The depressed piece of bone was removed by trephining. The dura was intact and pulsated normally. It was not incised. This operation was performed four days after his original wound.

A week after the operation his temperature was normal: there was weakness of the right face with deviation of the tongue to the right side. There was no weakness or anæsthesia of the upper or lower limbs. The knee-jerks and ankle-jerks were brisk and equal; the plantar reflexes were both flexor in type and the abdominal reflexes were normal on both sides.

He had no word-deafness nor word-blindness. He understood and executed verbal requests smartly, also a written order to put out his tongue.

He spoke with some difficulty. When asked his name he said: "Duncan M-M-M-M-enzies." He could not name a postcard, a pin, or a safety-pin. He called a fork a knife, but at once recognized his mistake, and when shown different objects, including a knife, fork, and spoon, and asked: "Is that a

knife?" always said "Yes" when the correct object was indicated, and "No" when it was not so. He wrote a postcard to his parents, writing their address correctly, but repeating several words. His message ran: "I am getting only, only, yours, Duncan."

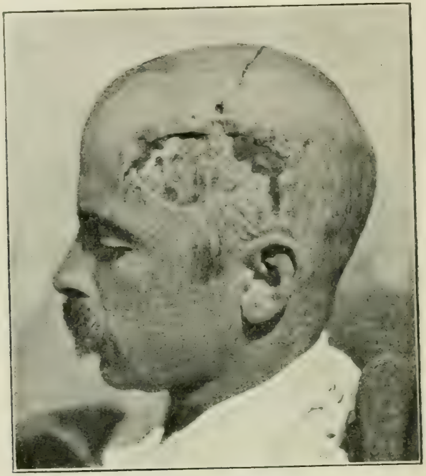

FIG. 3.—Left inferior frontal wound.

A week later he spoke better, but slower than normally, occasionally confusing his syllables. He called a coin a "Greek fenny," and a safety-pin a "sapety-pin."

A week later he no longer jumbled his syllables, and, save for a certain amount of slowness, his speech was normal and fluent.

This again is a fairly straightforward case of psycho-motor aphasia. Dr. Head might call it verbal aphasia or nominal aphasia, or both.

Case 4.—W. Y., aged 19. Temporal lesion.

He came into hospital without any available history, but he had probably been wounded four or five days previously at Gallipoli and brought straight to Malta, where I happened to see him.

There was a superficial entry-wound three inches vertically above the tip of the left pinna and an exit-wound half an inch above the posterior end of the left zygoma (fig. 4). The cranium was not fractured, and X-rays showed no abnormality.

He had no weakness or anæsthesia of the face, trunk or limbs. The reflexes, deep and superficial, were normal and equal on the two sides. There was no limitation of the visual fields.

He understood and executed simple verbal requests, but very easily became confused, e.g., when told to place his right hand on his left ear. He read aloud slowly and accurately, but did not understand what he read, nor did he

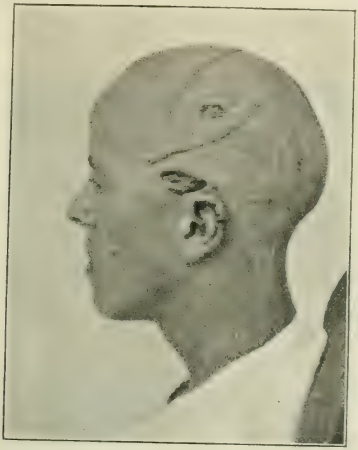

FIG. 4.—Left temporal wound.

make any attempt to execute written requests which he read aloud. When shown the statement $\begin{array}{c}2\\ \underline{2}\\ 5\end{array}$, however, he said, "That's wrong."

He spoke slowly and with difficulty, but could name objects shown to him. When speaking he occasionally jumbled up syllables.

This is a case of psycho-sensory aphasia, with word-deafness, paraphasia and word-blindness. Dr. Head would probably call it a combination of nominal and semantic aphasia.

Case 5.—C. E., captain, aged 22. Temporo-occipital lesion.

Patient had a penetrating wound by a shrapnel bullet. The entry was above and behind left ear, over the posterior end of the first temporal gyrus (see fig. 5). There was no exit-wound and an X-ray showed the bullet lodged $\frac{1}{2}$ in. to the left of the middle line, just above the occipital protuberance, i.e., near the tip of the occipital lobe.

Four days after his injury he developed meningitic symptoms with general epileptiform fits. The wound was laid open and several pieces of in-driven bone were removed from the brain. The fits subsided a fortnight after his injury.

When examined three weeks after the injury, he had a voracious appetite, possibly attributable to his temporal lesion. The wound was soundly healed.

Five weeks after his injury he had no weakness of face or limbs, no anæsthesia, normal deep and superficial reflexes, but a right upper quadrantic hemianopia in both visual fields, due to lesion of the lower lip of the left calcarine cortex.

Fig. 5.—Left temporal wound, with bullet in left occipital lobe.

He understood and executed simple verbal requests, but had difficulty in executing complex requests, e.g., to place the right middle finger on the left ear. He executed written requests, wrote fluently, but occasionally made spelling mistakes, e.g., mis-spelt "Malta" as "Matla." He copied accurately from printed to written letters.

In speaking he had slight hesitation in naming objects but ultimately succeeded. He stumbled over syllables in words like "Gallipoli peninsula." He spoke fluently as a rule.

Here again the aphasia was of psycho-sensory type, with slight word-deafness and paraphasia, or mal-construction of words. Dr. Head would probably regard it as a case of generalized loss, not belonging to any one category.

Case 6.—J. C., aged 25. Temporo-parietal lesion.

Patient was admitted to hospital without any history, but with a recent operation-wound, apparently about a week old, on the left side of the head, including a trephine gap 2½ in. above and 1 in. behind the left external

auditory meatus (see fig. 6). There were also an entry and an exit-wound, one at the left parietal eminence, the other just below the centre of the left zygoma. Which was which, we could not decide.

He had no weakness of the upper or lower limbs; there was doubtful paresis of the right side of the face. He had no hemianopia on rough test and no cutaneous anæsthesia or analgesia. The knee-jerks and ankle-jerks were normal. The right plantar reflex was extensor; the abdominal reflexes were brisk and equal.

He had considerable difficulty in understanding and carrying out even simple verbal requests. When shown written commands he spelt out the letters correctly but made no attempt to carry out the instructions. He

FIG. 6.—Left zygomatico-parietal wound.

wrote with a pencil, writing his name spontaneously and copying from printed to written letters. When shown the statement $\begin{array}{c}2\\ 2\\ \hline 5\end{array}$ he did not recognize anything wrong.

He could not repeat words dictated to him nor could he write to dictation even simple words like "hospital."

When shown scissors, he could not name them, but said, "I know what they are for," and made scissor-movements with his fingers. With a pencil, he said, "I know what it is," but could not name it.

A week later the word-deafness was less marked. He could not write to dictation but made mistakes in spelling, e.g., "arterly" for "artillery." He could repeat words dictated to him but could not name objects, such as scissors or a pencil, saying, "I don't know the name of it, I forget."

This case would probably be regarded by Dr. Head as being of the "nominal" variety. But on clinical and anatomical grounds it is a straightforward case of psycho-sensory aphasia.

I might have quoted many other cases of more profound aphasia, due to widespread lesions, but the cases which I have related have been selected because of the relative simplicity of the brain-lesion in each case.

We find then, in cases of psycho-motor aphasia, or aphasia of expression, a lesion in the anterior or psycho-motor area of the brain, whereas in psycho-sensory aphasia, or aphasia of comprehension, the lesion is situated in the psycho-sensory speech areas, whether psycho-auditory or psycho-visual. In both classes of case there is always a certain amount of intellectual disturbance, the equilibrium of the speech areas being rendered less secure, just as a table becomes unsteady, whether one of its anterior or its posterior legs happens to be broken.

We are familiar with Pierre Marie's view that all aphasia is merely an intellectual deficiency, due to disintegration of some part of Wernicke's zone. He claims that the essential fact of aphasia, of whatever variety, is insufficient comprehension of speech. We also know that Marie discards a special motor centre for speech and denies to Broca's convolution any special rôle in the function of speech, except in so far as it contains motor centres for the lips, tongue and larynx. He explains motor aphasia as mere intellectual deficiency *plus* anarthria, and refers this anarthria to a lesion of the lenticular zone (comprising the lenticular nucleus and its surrounding white matter).

But motor aphasia is not a mere paralysis of the lips or tongue; it is a true apraxia, due to amnesia of the movements of articulation. Moreover, if articulative difficulty were the cause of the speech loss, it ought to render the articulation of all words difficult. Now we know that most aphasics, however profoundly speechless, are not wordless. They usually have a "recurrent utterance" of some sort, and this recurrent utterance is articulated perfectly clearly.

It would, therefore, seem that the psycho-visual, psycho-auditory and psycho-motor speech centres do exist, and that lesions of these centres, disrupting the cerebral mechanism of speech, are the cause of our well-defined clinical types of aphasia.

Dr. Head by a series of ingenious and elaborate tests has arrived at a new, useful and philosophical basis of classifying aphasias. But this

does not necessarily mean that other classifications may not also be correct. Just as one observer may classify a crowd of people into men, women and children; whereas another may classify them into Englishmen, Scotsmen, Irishmen, Welshmen and foreigners; or another again into soldiers and civilians. It all depends on the angle from which the facts are viewed. Dr. Head has opened a new window into the chamber of aphasia. But this does not compel us to close up all the other windows, especially the anatomical and pathological windows, through which so much light has been admitted in the past.

Dr. Kinnier Wilson: It is with a certain amount of hesitation that I venture to express my views in regard to Dr. Head's recent contributions to the difficult subject of aphasia. Perusal of his papers is not enough, perhaps, to justify criticism or even to arouse full appreciation; it is evident that the student of the question must himself utilize some or all of Dr. Head's ingenious tests for the aphasic in order to be in a better position to pass comment on his conclusions.

Nevertheless, certain general considerations seem worthy of a little attention.

It is clear, in the first place, that Dr. Head's contributions must be viewed solely from the clinical standpoint. The question of localization is not raised, with the exception that he speaks throughout of "unilateral lesions," presumably in the left hemisphere. As there is no record of any pathological investigations, one cannot even be sure that the lesions have been strictly unilateral. Incidentally, I cannot agree with him that the "G.S.W. head" type of case is better for the study of the subject than vascular cases, for he himself admits that gunshot wounds tend to cause greatest damage on the surface of the brain, in addition to which there is the factor of commotio in all such patients. The fact that small hæmorrhages may catch projection fibres where they converge or diverge, is, in my opinion, much less important for the subject of aphasia than that association—i.e., transcortical, fibre systems should be involved, and these are obviously more likely to be impaired in cases where the damage is mainly cortical. The ideal case is neither the tumour nor the gunshot wound case, but that with isolated vascular lesion, the exact position of which there is opportunity to establish post mortem. Such cases are few and far between, but that increases their value when they are obtained.

Ignoring, however, the pathological side of the question, and the whole matter of localization, one may attempt to form some judgment on the clinical side of Dr. Head's work.

(1) We must be in agreement with him when he shows with clarity that the various types of aphasia are produced by dissociation of a definite mental process, which he designates symbolic thinking and expression. No one can suppose that aphasia is due to loss of motor or sensory power; if a patient has his speech musculature paralysed he is anarthric, or if paresed, dysarthric, but these are phenomena of a lower (middle) level, and the same is true of ordinary cortical blindness or deafness; these are not elements in aphasia. Dr. Head holds, further, that aphasia is not due to "diminution of general intellectual capacity," as Dejerine and others have also held in opposition to Pierre Marie, and some of my own cases may certainly be taken to bear this out. Not a few strictly limited cases of aphasic disturbance are on record to substantiate the truth of the statement. It appears to me, nevertheless, that some of Dr. Head's tests are tests of intellectual capacity, or at least of eupraxia, rather than of speech activity in the ordinary sense; in particular, his mirror test. Accepting Dr. Head's evidence from a large number of his cases of aphasia, that the patient has difficulties in imitating movements made by the examiner when the latter faces him, but can perform them easily when they are seen in a mirror, I am not able to follow the suggested explanation, that in the former group the imitative movements are "accompanied by more or less internal verbalization," whereas in the latter "no verbal symbol intervenes between the reception of the command in gesture and its execution." It seems to me largely a question of automatism. All of the patients are certain to have performed movements in front of a mirror every day of their life, so to speak, e.g., in shaving, hair-brushing, &c., and such types of movement become automatic; whereas, I do not suppose a single one of them had ever practised direct imitation movements. Their difficulty, therefore, I take to be one of practice and not of internal verbalization, and this is borne out by Dr. Head's statement that many improved very rapidly in this faculty. The test can, I think, be fairly set down as one of "glegness in the uptak" which varies enormously with the individual. Besides, not a little evidence is accumulating to suggest that automatic processes, automatisms, are located in the *right* as opposed to the left hemisphere. It may be remembered that Hughlings Jackson himself held that the right was the more "automatic" of the two; hence, one should not expect the same impairment of acquired automatisms in *left* hemisphere lesions, where one may presume the latter are located in Dr. Head's cases. The point, however, can only be alluded to in passing.

Again, the author maintains that aphasic defects are not due to destruction of images, showing by examples that many aphasics have not lost their images at all. I do not think the common view of aphasia is that in its different forms different types of image are necessarily *destroyed*, but that the patient is not able, owing to his organic disease, to awaken them when he wishes to. Thus a patient of my own after a stroke became absolutely mute. His state was one of complete motor aphasia; he had no words, not even "yes" or "no," and had apparently lost all his motor images, but on the night of the destruction of the Zeppelin at Potter's Bar he was a spectator of the wonderful sight and called out "Hallelujah," which word he has never since repeated in spite of innumerable efforts. The "engram" could not have been destroyed, and while it may be impossible as a rule to prove that images are not destroyed, for the practical purposes of speech they are "lost," and it seems to me to matter little, in a way, what has really become of them in such cases. Probably the question is largely one of the extent of the pathological lesion, and as in the majority of instances images can be shown to be retained, the lesions cannot be "knock-out" ones. After learning that in Dr. Head's strongly expressed view the affections of language from (presumably) unilateral lesions "are not based on a destruction of images," one is a little surprised to be told subsequently that he expressly excludes cases of aphasia where the patients "showed evidence of definite destruction of sensory images." It is admitted, then, that some cases are image-less. Of course, in the mind the concept of an object, say an orange, is so compact of differing linked images that it can be aroused in consciousness by stimuli from one sense avenue when others fail; Dr. Head has well shown in one of his routine tests how an aphasic can recognize an object by touch, say, when visual images fail him, and so on. Only in the most widespread cases of cortical impairment, therefore, would one expect destruction of images, and since the sensory components of the synthetized complete idea of an object are, I hold, located in widely separated cortical fields, it naturally follows that destruction of images on any wholesale scale must be a rarity. For example, too many cases have been classed as agraphia because the patient cannot write with his right hand as a result of a cerebral lesion, but too rarely is the test made with the left; for that matter, an agraphic patient, so-called, might conceivably still be able to write with the pen in his teeth, or held by his toes.

(2) This leads to a second consideration, which is, that Dr. Head holds that the familiar terms "aphasia" itself, "alexia," and "agraphia,"

should never be employed, because the disorders of language produced by a unilateral lesion of the brain are never "*exclusive* affections of speech, reading, or writing."

In another place he says—and I wish to draw special attention to the statement—the function which is disturbed by such lesions of the brain "cannot obviously be comprised under the headings of speaking, reading, and writing; for not only may the loss of power to carry out any one of them be partial, but the disturbance extends beyond their limits and affects other mental activities."

Now it may be granted at once that "pure" cases of these disorders are doubtless rare, that mixed varieties are the rule; and, for the sake of argument, one may say with Dr. Head that "inability to write is always associated with some other loss of function." It is, nevertheless, a clinical fact that one or other defect, in speaking, reading, or writing, may be *by far the most prominent* feature of a given case, so that while, for example, speaking and reading may be for practical purposes intact, writing may be all but impossible, as in a good case published in 1903 by Stanley Barnes, and similarly with alexia (combined with hemianopia) from lesions in the distribution of the posterior cerebral artery, as in a striking case of my own, and with pure motor aphasia as excellently shown by Dejerine. Rarity, even absence, of "pure" cases does not seem to me a conclusively adequate reason for abandoning useful clinical descriptive terms; especially since no one can deny the occurrence of difficulties in speaking, reading, or writing, and Dr. Head's own cases, needless to say, exemplify these admirably. If the absence of *separate* implication of any constituent in speech is to be advanced as an argument for the relinquishing of the above terms, exactly the same argument would apply to Dr. Head's own new classification of the varieties of aphasia into "verbal," "nominal," "syntactical," and "semantic" groups, since he himself admits that in most cases "two or more of these aspects of symbolic formulation are affected," and, again, "these terms are not intended to define the limits of the disturbance." In other words, since "pure" cases of these varieties never occur either, the argument for the abandonment of agraphic or alexic varieties on the ground of their never occurring in a "pure" state falls to the ground.

(3) If, now, we take Dr. Head's new subdivisions, we learn from him that in verbal aphasia the essential defect is one of word-formation; "words are evoked with difficulty and the vocabulary is greatly restricted; enunciation is slow and halting." In addition, writing may

be extremely difficult or almost impossible, and in any case "tends to show the same errors as articulatory speech." In nominal aphasia the patient has difficulty in naming letters, words, or objects. In addition, however, "writing is gravely affected," and the power of reading is greatly hampered. The third group, syntactical aphasia, is one where the patient talks jargon. Here also, however, "patients suffering from the more severe degrees of this affection cannot write a letter," and we are told that the disorder "is essentially one of auditory balance and rhythm." Finally, in semantic aphasia there is a want of recognition of the full significance of words and phrases. The patient "fails to comprehend the final aim or goal of an action"; he cannot "formulate symbolically a general conception."

Of the clinical occurrence of these types there is, of course, no question, and we owe a debt of gratitude to Dr. Head for the emphasis he has laid on them. Their content and significance have never been so closely examined and so admirably illustrated. In particular, it appears to me sufficient attention has never been paid in this country to the grammatical aspect of aphasia, and as I personally have long been aware of its importance and have much material bearing on it, I should like to take this chance of giving my support to Dr. Head's conclusions as far as this part of the subject is concerned.

At the same time, I do not see that the "new" types, so-called, in any way *run counter to* the hitherto accepted and familiar subdivisions. Verbal aphasia closely resembles ordinary motor aphasia; the nominal variety is one commonly observed, and I may say incidentally that it is in my opinion capable of sufficiently close localization. The neurologist is aware of the association between Mills' naming centre as it has been called, and integrity of function of the upper temporal lobe, and I have seen more than one excellent example of this defect in naming from strictly limited lesions in that part of the brain. Dr. Head includes much more than naming defects, however, in his nominal aphasia, as we have seen, though this naming defect is the special clinical feature. Syntactical aphasia, again, with its jargon-aphasia, has been described often enough in the group of sensory aphasia, and I showed a perfect example of this variety at a clinical meeting of the old Neurological Society. It will be remembered that, as already noted, Dr. Head agrees the disorder is essentially one of *auditory* "balance and rhythm."

The fourth classification leads me to what I think the most significant part of the new doctrine. Perusal of Dr. Head's illustration of semantic aphasia shows that it is largely identical with agnosia, in

particular with that described by Liepmann and others as ideational agnosia. The examples furnished make this quite clear. Similar illustrations were given by myself from personally observed cases in my paper on apraxia.

Taking aphasic symptomatology as a whole, it appears to me that what Dr. Head has done is to make a new division which may be said in some ways to run across the classical divisions. Thus instead of speaking of agraphia and alexia, he speaks of verbal, nominal, and syntactical aphasia, but he admits that what is called agraphia or dysgraphia is a feature of all three, while alexia occurs at least in the second, if not in others. This again brings out the point to which I have referred above, that there is not, indeed there cannot be, any *subversion* of previous clinical knowledge in Dr. Head's work.

Dr. Head has not, as far as I have observed, used either of the terms agnosia and apraxia in his papers, obvious though it is that he is frequently describing apraxic or agnostic phenomena. Hence the significance of the remark already quoted from his paper, that in aphasia the disturbance extends beyond speaking, reading, and writing, and affects other mental activities. I believe the subject can best be understood if disorders of these "faculties," and the other mental activities mentioned by him, are looked at from the point of view of apraxia and agnosia. I do not wish to take up the time of the meeting further, except to sketch in a word what line I follow. I accept the general division of aphasia into receptive and executive defects ; I hold that ordinary motor aphasia is but a part of motor apraxia, ordinarily called apraxia, and sensory aphasia of agnosia. The patient with auditory agnosia hears sounds, but does not recognize them, i.e., does not know their meaning. Among these sounds may be word-sounds, which he hears, but the content, the symbolic meaning, is for him unknown. The same is true of alexia as a variety of visual agnosia. Defects of this sort may certainly occur in unilateral lesions of the left hemisphere. On the executive side motor aphasia is a part of apraxia ; the patient is not paralysed, but he cannot say what he wants to say, as the apraxic cannot do what he wants to do.

The analysis may be carried closer, and the clinical varieties of aphasia may be shown to have their counterpart on both executive and receptive sides in the matter of the "faculty" or "psychical activity" of music, and in the reckoning faculty. My view is that on the clinical side, which is all that for the moment concerns us, speech disorders, just as reckoning and music disorders, are parts of the wider impairments of function known as apraxia and agnosia.

Dr. Stanley Barnes (Birmingham) remarked upon the very interesting way in which Dr. Head had started what appeared to be a new form of investigation of disorders of speech. He thought the scheme of examination which Dr. Head had put forward was likely to lead to more accurate results and a clearer understanding of the speech mechanism than those hitherto obtained. He himself had not had experience in examining patients on this new basis, but thought that the method now suggested was likely to carry them as much further in the analysis of speech defects and localization of speech function in the future as Dr. Head's extremely valuable researches into the various forms of sensation had enlarged their knowledge of the sensory paths. At the same time, he was rather sorry Dr. Head had published his paper in the somewhat incomplete form in which it had gone out. It would have been better if he had either included with it as published the addendum he had just read out, or waited a little longer before publishing. It gave the general impression that, after all, localization of speech was a thing which had now practically gone by the board. He did not for an instant believe that was Dr. Head's intention. He hoped that would be made clear in Dr. Head's reply.

A point he would have liked to have heard more emphasized by Dr. Head was that intellectual faculties were so closely bound up with speech that one must expect them to be represented in the same area of the brain, and that any serious defect in speech must mean a serious defect in intellect too. In some of the cases Dr. Head quoted there was a suggestion of great speech defect with no material loss of intellectual capacity; he, the speaker, found it difficult to believe that. The neurologist was dealing with patients of such varying degrees of intellectual capacity and he knew so little at first hand about the patient's intellect as it was before his lesion, that it became very difficult to estimate what was the degree of intellectual defect, if any. It was his own belief, from clinical and psychological investigation, that the two functions, speech and intellect, were largely interdependent and were represented in the same area of the brain.

It might be going further than Dr. Head wanted to go that night, but he (the speaker) would like to hear whether Dr. Head could suggest any localization, or any element in localization, with regard to varieties of speech defects. His own opinion, which he had held for several years past, was that the areas which had been hitherto suggested were too much restricted. He thought the whole mass of the information which had been received had been argued about on too narrow a basis, because

the localization of the various functions had always been assumed to be too microscopical. Thinking out the matter on purely psychological grounds, knowing something of the localization of the motor function of the brain, and adopting Hughlings Jackson's views as to the representation of particular areas, he could not believe that such an important function as that of speech could be localized in any small area of the brain: it must require a vast representation. He believed that the area of the left side of the brain devoted to speech stretched from the foot of the third frontal convolution in front to the angular gyrus behind; and from the lower limits of the supramarginal convolution above to the middle and probably the inferior temporo-sphenoidal convolution below. This large area, including most of the island of Reil, he believed to be mainly devoted to speech (both internal and external) and intellect. He believed there were no sharply-divided compartments in this area where it could be said that this or that part of the cortex was solely devoted to particular elements of the speech function, but he did believe that in the main the hinder end of this area was mainly one in which visual impressions were received and elaborated into such a form that they were ready to be received in consciousness and interpreted as speech; and that in the same way the temporal convolutions were mainly devoted to re-representing auditory impressions as speech; whilst the frontal end was chiefly concerned with the emission of speech in the form of words spoken and written.

Dr. Head had told of a variety of lesions and a variety of results. There was a suggestion—nothing more—that whether there would be one type of aphasia or the other would be determined according to the intensity of the lesion, and not according to its localization. In this matter his experience was similar to that of the last speaker and others who had spoken that night. He thought there could be no doubt that a lesion of the hinder end of the speech area would give different results from those of a lesion in the fore end. But he thought that in all these cases there would be a depression of intellect, that the whole intellectual faculty would suffer to some extent in whatsoever portion the speech area was damaged. And he agreed with Dr. Collier that the more sharply defined was the type of aphasia, the more certainly would there be a subcortical as well as a cortical lesion. He did not think there was any sharp line of definition between motor and sensory cortex, but that they were intimately intermixed, and that as one passed to the fore end of the brain it became more motor, and as the occipital end was approached it became more sensual (visual),

while on reaching the temporal lobe it was mainly auditory in type. He did not doubt that Dr. Head had explained his own cases, in the main, correctly. But, there again, he agreed with Dr. Wilson that those cases were not, essentially, any better for the purpose of determining localization than were the types members had seen. He had seen cases of bullet wounds in which the brain had been exposed by operation, cases in which the missile had crushed through the dura and those which had suffered concussion effects through a slanting blow, and it had been difficult to define the limits of the lesion, either in the surface direction or in depth. He thought Dr. Head would agree that, though it was clear that the lesions in his cases were mainly cortical, yet, to a considerable but varying extent, they might be subcortical.

He did not yet know exactly what Dr. Head meant in regard to the type of theory of which Sir James Purves Stewart spoke. He (the speaker) thought the main question at issue as between neurologists to-day was as to whether there was an intermediate cortical "centre" through which impulses passed, whether they were elaborated as coming from the sensory side and were coded as they were passing out in the emissary form. He agreed that there was, as yet, no absolute proof that the visual impulses did not go directly into the main mass of the speech area, that they were entering into intelligence in such a form that one could say that at such a particular place they had been re-represented or integrated to a higher stage, so that they could then reach consciousness. But he felt very little doubt that the process of integration was going on; he thought it was operative in an area not sharply defined from the remainder of the speech area, and that neurologists could use such a localization as had been worked out in the past very valuably from the point of view of localization of speech function.

J. HERBERT PARSONS, F.R.C.S.[1]: In his two admirable lectures on aphasia Dr. Henry Head has not defined precisely the psychological aspects of the question. Doubtless these aspects were fundamental to his thesis and were ever present in his mind, though the exigencies of the circumstances prevented their exhaustive discussion. The fact that he has laid so much stress on "meaning," as shown by the introduction of the term "semantic aphasia," is ample evidence that the omission is not due to oversight; it proves, in fact, that accurate psychological principles are the very foundation of his inspiration. It

[1] These remarks on "The Psychology of 'Meaning' in its Relation to Aphasia" were handed in during the discussion but were not actually delivered.

is, however, regrettable that these aspects have not been treated more definitely; in the first place, because they afford the most valuable support to his views; and in the second place, because their omission is likely to lead to misconception. Indeed, there is reason to think that such misconception has already arisen. It is readily explicable, for neurologists most interested in the subject naturally view it from the anatomical and physiological standpoint, rather than from the psychological, owing to their training and clinical experience. They are therefore liable to ignore or misconceive the exact rationale of pathological anatomical lesions in the genesis of disorders of the highest psychic activities.

The emphasis laid upon the semantic aspects of aphasia is the sign of a great advance in our conception of the disease, an advance inspired by the immortal work of Hughlings Jackson, confirmed by the previous researches of the distinguished author of the lectures, and founded upon the psychological aspect of the subject. Sound scientific psychology is therefore the crux of the question, and has hitherto not received the attention it deserves.

The psychology of "meaning" has been curiously neglected, and it is only of recent years, largely owing to G. F. Stout and the biological psychologists, of whom Lloyd Morgan is the outstanding example, that it has been analysed according to scientific principles. The work hitherto accomplished in this field is almost limited to the lower psychic activities, especially at the perceptional level. But "meaning" from the biological and psychological points of view has undergone profound phylogenetic and ontogenetic evolution, and speech is the symbolic manifestation of the highest and most complex form of "meaning."

At the lowest biological level, where the reflex arc constitutes the whole nervous mechanism, we can only conjecture the conscious accompaniments of nervous activity. It is probable that it is a vague undifferentiated sentiency, possessing a maximum of affective tone and a minimum of cognition. To it the term "meaning" is scarcely applicable, for the response is little more than a tropism: yet it would be unwise to deny the presence of a plus or a minus affective tone—pleasure or unpleasure—and this is the primitive germ of "meaning." In the higher species, in accordance with a very general psychological law, the conscious accompaniments of the reflex act have been suppressed.

At a slightly higher biological level, reflex arcs have become co-

ordinated and integrated, and the response assumes the character of instinctive reaction. It is unnecessary to labour over the conscious accompaniments of the inceptive stage of the instinctive level, for it passes by imperceptible gradations into the perceptual level which is the summit which most lower mammals have attained.

At the perceptual level, in any given first experience, sensations are aroused in the organism by physical stimuli from the outer world. Psychologically, as an impression of the given situation, sensations are objective. They undergo differentiation and subjective selection. The selected items are then reintegrated. The reintegration is of the nature of a truly creative synthesis, and issues in the form of an entirely new order of consciousness. To resume in other words, the relatively undifferentiated psychoplasm—to use a useful, but awkward and too concrete term—is differentiated into specialized affective and cognitive elements, which are reintegrated, thus undergoing a synthesis which is the "meaning" of the given experience. Perceptual "meaning," suffused with affective tone, issues in instinctive conative activity. This itself by "backstroke" supplies new modifying stimuli to the already altered psychoplasm. For, in addition to the satisfaction or unsatisfaction of the successful or obstructed instinctive reaction, the motor activities are accompanied by somatic afferent impulses modifying the cœnæsthesis which forms an important part of the conscious state.

Thus, at the end of the completed reaction the "meaning" has become enriched and complicated on the affective side by the impact of the afferent impulses derived from somatic and visceral sources, and on the cognitive side by the results of the conative activity. This altered "meaning" is stored up, and, though depressed below the threshold of consciousness, is capable of being revived and recognized on the occurrence of a cognate experience.

On the next occasion of a similar experience, in addition to the factors of the first occasion are the factors derived from recognition and revival of this concrete event which has been lived through. Learning by experience is the utilization of these revived factors, and consists chiefly in the addition of cognitive elements. The assimilation, differentiation, subjective selection, integration and synthesis of the already more plastic psychoplasm results in a higher, more complex type of "meaning"—a "meaning" which we recognize as of higher grade and explain as an intelligent modification of the hitherto essentially instinctive reaction.

In the human organism the instinctive elements, as well as innate

dispositions of a higher order, are already present in immature form as inherited characteristics. The simpler syntheses of the lower mammal are complicated by this wealth of innate factors. Not only is the first experience thus complicated, but each succeeding experience is further complicated, and experiences which are only remotely similar add their quota to the already complex mass of factors, inherited and acquired. Thus, even at the perceptual level "meaning" becomes extremely complex.

With the evolution of free ideas, and still more under the fully developed assimilation and apperception of conceptual thought, the complexity is further increased.

Hitherto no special stress has been laid upon the social environment of the individual, but we have now learnt that man is a "socius" from very early infancy. Long before a child can talk the "meanings" of the different phases of his experience of the outer world acquire characteristics which are imprinted upon them by the assimilation of special qualities derived from intercourse with his fellow creatures, of whom, of course, his mother is the most important. Though this assimilation should be regarded as a perpetual recurrence of the cycle of differentiation, subjective selection, integration, and creative synthesis, the ultimate results are equivalent to an interaction of old and new "meanings," resulting in an infinity of still newer, richer and more refined "meanings." At this stage conative activities themselves assume a synergy at a higher level, which is the dawn of true volition. The synthesis of the child's mental processes into a "self" is the product of his social intercourse, and his conative activities now show a projicience hitherto absent; to the outside observer his gestures are no longer merely passive signs of his mind's activities, but active indications of his feelings and desires. This is the dawn of language.

It would, of course, be a gross error to imagine that this brief sketch gives any true idea of the complexity of the evolution of "meaning." There is another aspect which is particularly illuminating from the point of view of aphasia, especially in association with Dr. Head's work. The analogy between the primitive undifferentiated instinct—with its "all or none" response, comparable to the "mass-reflex" of the isolated spinal cord—and protopathic sensation, has been pointed out and exhaustively elaborated by Dr. W. H. R. Rivers in his "Instinct and the Unconscious." Similarly the differentiation of the primitive instincts, with the partial suppression of the vague elements belonging to the protopathic level, is comparable to epicritic sensations.

These essentially biological and physiological aspects form the foundation of the more purely psychological aspects already considered, and are of the greatest importance in the elucidation of the phenomena of aphasia.

Enough has been said to show that "meaning" at the speech level is extraordinarily complex. Yet the methodology of analysis is now taking shape, and its application has already elucidated many of the obscurities at the perceptual level. Much yet remains to be done at higher levels. Even when this is done there yet remains the enormous field of the psychology of the concrete individual, a matter of no small importance in dealing with aphasic patients of different degrees and varieties of nature and nurture. The "meaning" of a given situation will differ for different individuals according to their inherited instincts and innate dispositions as well as their acquired experience, especially that derived from their social environment. These differences will manifest themselves in their motor responses, and above all in speech.

I have hitherto considered only the organization of thought, using that term in a wide sense. The difficult question how far the organization of conceptual thought is dependent upon speech—"internal speech," in Dr. Head's terminology—has recently received considerable attention (*British Journal of Psychology*, General Section, October, 1920, pp. 55 to 104). Certain it is that some re-synthesis occurs in the symbolization of thought which is the essential feature of speech, and that a further re-synthesis occurs in the transformation of internal into external speech. Light is thrown upon this part of the process by the study of the development of speech in the child and particularly by the psychology of reading. One elementary fact is demonstrated by the latter study, viz., that the grouping of concepts gradually develops a wider span—from the reading of letters, or at any rate monosyllabic groups of letters, the child progresses to the apprehension of groups of words. I have dealt with this portion of the subject elsewhere (*British Medical Journal*, August 22, 1914).

When it is realized that the neurologist attacks the problem at the most complex stage of its development, and that he is confronted with focal anatomical lesions which are difficult to reconcile with the psychological complexities, it is not surprising that his explanations should be crude and of only partial validity. Much of the crudity is due to the unfortunate prevalence of the term "centre." So long ago as 1904, speaking of various pupillary "centres" which have been described, I wrote: "One is inclined to wish that this term might be abolished

from neurology, so great is the ambiguity and confusion to which it has given rise. The intercommunications between the cells of the central nervous system are so complex that there are almost innumerable alternative paths whereby any given group of efferent cells may receive excitatory impulses. Our aim should be, first to determine the efferent cells governing any given movement, second to determine as far as is possible which are the afferent tracts which can lead directly or indirectly to those cells, and third to find out *how* each of those afferent tracts leads to the efferent cells. The last problem is the most difficult, and we shall find that many of the afferent tracts are very devious, and have many cell-stations on their way. Each of these may be dignified with the title 'centre,' or better, none of them" ("The Neurology of Vision," 1904, p. 50). The term—and the train of thought associated with it—is eminently unsuitable when dealing with the higher psychic phenomena, such as "meaning" and the speech which subserves it. Scientifically, the unity of the mind is correlated with a synthesis of nervous activities which are so widespread throughout the nervous system that the term "centre" is ludicrously inadequate. Yet these syntheses are dependent for their perfection upon congeries of factors which are in some degree accessible to analysis. In disorders of speech for instance, lesions on the afferent side, of which the auditory are of prime importance, will cut off some of the pabulum for differentiation, subjective selection, integration and synthesis. Such cases have been described clinically as sensory aphasias.

Dr. Head has somewhat forcibly deprecated the use of such terms as motor and sensory aphasia, alexia, agraphia, anarthria, and so on, but his protest belongs to the order of counsels of perfection. That such terms have often been unwisely chosen and inaccurately applied may well be granted, but their use as descriptive terms can scarcely be avoided with any great degree of consistency. In studying a complex "higher level" subject, of which the fundamentals are very imperfectly known, the lumping together of allied factors under one name is not only convenient but unavoidable if awkward circumlocutions are to be avoided. Doubtless semantic aphasia is a form of agnosia: both terms have their uses as well as their abuses. It is a matter of precise definition and consistent adhesion to the defined significance.

The study of aphasia is such a pre-eminent example of the necessity for the collaboration of psychologists and neurologists and of the advantages which would accrue to both sciences by more intimate co-ordination that it is, I hope, a sufficient excuse for one so unversed in the clinical aspects of the subject having the temerity to make this communication.

Dr. HEAD (in reply) said he could not imagine a task more difficult than to be questioned by the various speakers and to have to answer them unprepared. He understood Dr. Collier to be in general agreement with the clinical aspect of what he (Dr. Head) had put forward. He could not understand why Dr. Collier said that in spite of the fact that the terms used were fundamentally incorrect, and "physiologically, anatomically, and psychologically impossible," he intended to keep to them. It was for such reasons he (the speaker) had been bold enough to try to replace them by something which would be more fundamentally correct, more physiologically adequate, more psychologically possible. In speech they were dealing with a high-grade psychical aptitude far above the level of sensation. This function demanded the integration of a number of other functions before it could be carried out, and in this way speech seemed to resemble sensation; either could be upset by lesions of the brain, which did not necessarily destroy the whole act, but destroyed it in part. The mere fact that speech could be disturbed in part showed that those areas of the cortex which were attacked by these various lesions were not all of equal value. He gathered from Dr. Collier that he considered most of the lesions which produced these disturbances affected either the entering or the emissary path—i.e., in the pure forms; but with lesions of the entering path they were not concerned with speech at all, but with a disorder of some aspect of sensation. On the emissive side such a lesion should produce a pure anarthria.

Proceeding to the discussion of syntactical aphasia, he would try to unite the two parts of Dr. Collier's statement together. In the first part of his discussion on syntactical aphasia, Dr. Collier had hit on the explanation he (the speaker) had given of its origin. If a man had taken too much alcohol his legs became ataxic; he struck against chairs and tables in his progression, and the repercussion on himself told him he had not proper control of his legs. But if at the same time his speech was disorderly, he did not learn this through his somatic sensations, he knew it because he heard his speech to be wrong. Syntactical loss was due to the fact that the man could speak, but had lost regulating power over the rhythm and balance of what he said. The actual words, like all words spoken in these disturbances of language, were representative of the parts not affected. He was glad to find that, in this matter, Dr. Collier and he had hit upon the same explanation.

He was sorry to see that, later on, Dr. Collier used the terms "visual speech" and "auditory speech," as he (Dr. Head) did not know what

they were; there was speech, but not visual speech or auditory speech. When a person was speaking, his actions were carried out by physiological processes above the visual and the auditory levels. In most cases of aphasia, image-formation was not definitely impaired. True, there might be a combination of loss of image-formation and loss of speech. One of the best cases on record was that which Stauffenberg worked out in v. Monakow's laboratory in 1913. This was certainly a case of aphasia combined with what Hughlings Jackson would have called imperception.

He would like to correct the statement that his patients were selected because the lesion was thought to be cortical; with these wounds no one could be certain whether a lesion was cortical or subcortical. His object had been to see what were the actual forms assumed by destruction of speech—i.e., a high psychical function—in consequence of various organic lesions. It was impossible to suppose that the relation between organic destruction and the loss of psychical function stood in the relation of so much loss of cortex to so much disorder of psychical function. The effect of an organic lesion of the brain on the functions of speech might be compared with the result on an electric light installation of destroying a varying number of the accumulators; the area of the brain supplied the vital force, which was the basis of the physiological energy which underlay this complicated psychical act. It might easily happen that it did not disintegrate, but rather lowered the general activity and vitality of the power of speech. As a fact, however, injury to one portion of the brain was more likely, under favourable conditions, to lead to the breaking up of speech in certain directions. But before attempting to decide the situation of the lesion which produced some specific disorder of speech, it was absolutely necessary to ascertain what the disturbance was. It was of no use trying to localize the position of an unknown function on the surface of the brain. That was why he had kept away from all questions of localization at present; he wanted first to determine what function was disturbed, before putting forward views as to what lesion was likely to produce it.

Most of the speakers had accused him of being an empiric. He was delighted to hear it. The whole point in his papers was that he wanted to follow the line of English empiric philosophy dating from Locke and passing into medicine through Hughlings Jackson. The latter said, in a famous passage, "Put down what the patient does get at, and avoid all such terms as amnesia, &c."

The next point on which he wanted to insist was that these functions were not faculties; one speaker used the word "faculty" three or four times. "Faculties" came from mediæval philosophy, and had now, fortunately, been got rid of in psychology. The use of such words as "general understanding," "aphasia," "amnesia," "memory" had been deprecated by Hughlings Jackson, who pointed out that there was no such thing as memory apart from things remembered, and no consciousness apart from conscious actions. The only possible way to find out what happened in consequence of an organic lesion was, not to use words such as "reading" and "writing" which were only *a priori* conceptual terms, but to observe what did actually happen. Had he been bold enough, he would have liked to have spoken of the "*x* group of functions," which broke up into the "*a*, *b*, *c* and *d* groups." But he dare not do that. He had been obliged to give them names, and it was those names which were going to lead him to perdition; because everyone would settle upon them and think they all meant something he did not intend; another set of conceptual terms would have been invented, to load up this unfortunate subject and obscure it further. There were no such things as "types" of aphasia. A vast group of functions were employed in the acts called speaking, reading and writing, and it was these which split up in certain ways to produce the clinical forms of disordered speech.

Sir James Purves Stewart simplified the question by dividing speech affections into psycho-motor or apraxia, and psycho-sensory or agnosia. It seemed to him (the speaker) that this was a matter of words; he did not know what psycho-motor meant, and apraxia was only a convenient term for certain aspects of defective cerebral activity. Neither did he know what "psycho-sensory" meant; the sensory side of speech must consist of sensations. When it was said that the psycho-sensory aspect of speech was agnosia, this was only saying in Greek that the patient did not appreciate the significance of impressions which came to him from without. With regard to the cases described, it was extraordinary how anybody, working under the conditions surrounding Sir James Purves Stewart in Gallipoli and elsewhere, should have been able to take such photographs and such elaborate notes. It filled him with admiration. But all these cases tended to have their value decreased because they were seen in the acute period, a period which in many cases extends over many months. It was difficult to say what specific form would be assumed afterwards by cases showing such a widespread lowering of vitality. Cases 2 and 3 appeared to have had verbal loss,

with difficulty in spelling. If they could have been watched over a long enough period, he thought they would be found to be verbal aphasics. No. 4 seemed to be too diffuse to be at all definite, but No. 5 was a severe example of nominal aphasia.

Dr. Wilson seemed to think that aphasia and apraxia could be put together under one heading. Both were convenient words if they were not carried to definition. The moment one drove "apraxia" to definition one saw it did not exist. There were forms of apraxic action, which no one had described better than had Dr. Wilson himself; but when one used the word "apraxia" to explain loss of speech, it did not seem to explain more than the most simple of aphasias. He wished to get rid of all such terms.

Dr. Stanley Barnes stated that the impression had got about that localization had gone by the board. If by this was meant the localization of "alexia," "agraphia" and "aphasia," he was glad he had given the correct impression. By localization he understood that affections of certain parts of the brain were liable to produce breakdown of this group of functions in one direction rather than in another. The anatomical basis of that form of localization he hoped to put before the Section on a future occasion. He did not believe in localization as it was ordinarily stated; he did not believe in the localization of what was called "motor aphasia," or "sensory aphasia," because he did not believe in the existence of motor and sensory aphasia.

With regard to the cortical or subcortical nature of the destruction, no one would suppose that gunshot wounds were other than gross lesions, and the wonder was that after the shock had passed off any of them were found to have produced specific loss of function. Dr. Barnes laid stress on a general intellectual defect; but psychologists to-day did not believe in general intellectual defects. This could be seen in the case of memory. Jackson said there was no such thing as general memory; there were only definite things remembered. Speech was a form of intellectual activity; therefore in saying there was a defect of those processes which were responsible for speech, it was assumed there was a defect of intellect. But this was a specific loss of function and there was not that gross widespread defect of intellectual aptitudes as was found in the slighest case of dementia.

Publications Recently Received.

Studies in Word Association. Under the direction of C. G. Jung, M.D., LL.D. Authorized translation by Dr. M. D. Eder. Pp. 575. London: William Heinemann, 1918.

Nearly half of this large volume is devoted to the study of the value of the response to the stimulus word. Jung and his co-workers have for this purpose investigated thirty-eight normal persons in great detail under varying conditions, and they have brought out interesting and in some ways unexpected results. As might have been surmised, they were able to show that on distraction whether external or internal, and in states of fatigue, the associations were of a more superficial kind than when these factors were absent; but a surprising result, obtained from the study of normal people, consisted in the fact that, other things being equal, educated people gave more superficial reactions than the uneducated. "The uneducated knows the word less as a verbal sign than as something with a meaning. Hence, single words are apprehended by them according to their meaning in some imaginary sentence; to the educated the stimulus word remains merely a word without any special meaning." From the different kinds of reaction which the experimenters classified, Jung has been able to make further progress with his psychological types.

To the psycho-analyst the main outcome of these investigations is that the importance of marked prolongation of reaction time as a complex indicator remains unimpaired. Though fatigue and external distraction may cause prolongation, they do not do so with the same success nor in the same selective way as the internal distraction of emotion.

In an elementary chapter Professor Bleuler seeks to demonstrate the fact of the unconscious. Unfortunately nearly all his proofs are for something of the nature of the fore-conscious or of a co-conscious which reasons after the same fashion as the conscious, the part of the subconscious which enables us by mental process to avoid jostling passengers in the street when we are thinking of something else. That is not a part of mental process which is in need of much demonstration. Its conception arouses no opposition, and it is probably accepted by everyone. What is needed is clear proof—and such is not difficult to find—of a subconscious whose contents never emerge into consciousness except by distortion, although they have great influence on our thoughts and actions.

There are useful chapters on word reactions in psycho-analysis and treatment, on the psycho-galvanic reaction and on associated physical changes.

It may be said that the work is one of great value, that it strengthens in every way the doctrines of psycho-analysis; for it is a record of facts derived from painstaking work, and is free from the speculative views which run through much of Jung's later work.

Dr. Eder deserves the highest praise for the translation; so excellent is it that the book might have been originally written in English.

The Psychology of the Special Senses and their Functional Disorders. (The Croonian Lectures delivered before the Royal College of Physicians in June, 1920.) By ARTHUR F. HURST, M.A., M.D., F.R.C.P. Pp. 123. London: Oxford University Press, 1920.

In these lectures Dr. Hurst gives an account of his observations on cutaneous sensibility, visceral pain, superficial reflexes and affections of the special senses, particularly in connection with the war neuroses. He accepts Babinski's definition of hysteria as "a condition in which symptoms are present which have resulted from suggestion and are curable by psychotherapy." He also agrees that all the ordinary forms of loss of somatic sensibility and limitation of the visual fields found in this disease are suggested to the patient: he gives instances to show, however, that in some cases during the war the suggestion came from a gross nerve injury. But in support of this view, with which all will agree who have had the opportunity of making observations on wounded soldiers, he cites cases where the "anæsthesia" occupied the whole area usually given by anatomists as the supply of the ulnar and median nerves. There is no note to show how this loss of sensibility was discovered. The usual form assumed in our experience by such hysterical conditions is for the whole anatomical area attributed to the injured nerve to be insensitive to prick, a condition which never occurs even after complete division of either the ulnar or median nerves. It is not an hysterical perpetuation of the condition produced by the injury but a superposed state due to suggestion by the medical examiner.

The author believes that the abdominal and plantar reflexes can be abolished in uncomplicated hysteria, although precautions are taken to protect the skin of the abdomen and the foot from cooling. He also considers that in the larger number of instances tenderness of the skin in cases of visceral disease is due to suggestions on the part of the observer.

He gives several cases of hysterical deafness and states that the auditory motor reflex may be absent and vestibular tests alone can show whether this condition is hysterical or not. Total blindness, even when accompanied by loss of reaction of the pupils to light, may be hysterical and undergo rapid cure.

Festschrift zur Feier des 70 Geburtstages von Hofrat Professor Dr. H. Obersteiner. Edited by Dr. OTTO MARBURG. In two parts, pp. 502. Leipzig and Vienna: Deuticke, 1917-18.

Professor Obersteiner attained his 70th birthday in 1917, and these two volumes were compiled by his pupils in his honour. They contain sixteen papers on a variety of neurological subjects, such as concussion of the spinal cord, gunshot wounds of the visual centre, psychoses and neuroses of war, loss of sweating with lesions of the spinal cord, nervous disorders of hearing due to explosions, and a beautifully illustrated paper on encephalitis lethargica. They form a worthy tribute to the eminent man to whom they are dedicated.

Arbeiten aus dem Neurologischen Institute an der Wiener Universität. Founded by Hofrat Professor Dr. H. OBERSTEINER. Edited by Professor Dr. OTTO MARBURG. Volume xxi, parts 1 and 2, June, 1914; part 3, February, 1916. Volume xxii, part 1, July, 1917; parts 2 and 3, October, 1919. Volume xxiii, part 1, October 1920. Leipzig and Vienna: Deuticke.

This well-known series of Studies from the Neurological Institute, founded by Professor Obersteiner in Vienna, appeared intermittently during the War under the editorship of Professor Otto Marburg, the present director.

The volumes which have recently come to hand contain papers on the anatomy and physiology of the vestibular nerve, the development of the cortex cerebri, several studies of the structure of the cerebellum, together with a long account of the comparative anatomy of its nuclei. Other papers deal with the relation of multiple sclerosis to syphilis, the pathological histology of the cerebellum in general paralysis, and of encephalitis lethargica. Recent experience of gunshot injuries is responsible for "Auto-regeneration of the Peripheral Portions of Severed Nerves," and "Pathology of War Injuries of the Spinal Cord." The editor gives a short account of a case of amnesia and of the structure of the Pineal Gland.

Military Psychiatry in Peace and War. By C. STANFORD READ, M.D. Pp. 168. London: H. K. Lewis and Co., 1920.

This volume is recommended to psychiatrists, psychologists and neurologists as presenting on the whole a reasonable biogenic view of the development of the graver mental disorders of soldiers. It is written by a psychiatrist of wide experience who has been led through modern psychological investigations and a realization of the inadequacy of the pathology advocated by the older alienists to recognize a psychogenic origin of many of the psychoses.

Between August 1914 and May 1919, 12,320 patients suffering from mental disease passed through "D" Block, Netley, which was the Clearing Hospital

for such cases from abroad. Of these, 331 were officers and 11,989 N.C.O.'s and privates. For a considerable period of the war the author was in charge of the psychiatric wards at Netley, and 3,000 patients passed through his hands. He was able to follow up the subsequent histories in the majority of cases, and in consequence was in a position to test his view that mental conflict is the most important ætological factor in the production of war psychoses.

One of the interesting and possibly far-reaching conclusions to which he has arrived is that too much stress has been laid upon exhaustion and alcoholic excess as causative agents by English and French observers. These factors in dementia præcox, paranoid, confusional, and manic-depressive states, &c., must be relegated to a position of secondary importance since they appear to be potent for harm only in so far as they aid the deleterious effects of mental conflict. This he believes to be motivated in the psychoses mainly by the sexual instinct and not by the instinct of self-preservation as in the psychoneuroses.

The largest number of his patients came under the groups of dementia præcox (20 per cent.), confusional insanity (13 per cent.), and mental deficiency as such without superimposed psychoses (13 per cent.). The prominence of paranoid tendencies in the early phases of all psychotic disorders in soldiers is noteworthy, and is to be explained as a direct consequence of the training and environment of the soldier and the liability for the manifestly abnormal individual to be the butt of his companions.

The absence of an index is largely compensated for by the arrangement of the material in short chapters.

Standard Method of Testing Juvenile Mentality by the Binet-Simon Scale and the Porteus Scale of Performance Tests. By NORBERT J. MELVILLE. Pp. 157. Philadelphia and London: J. B. Lippincott Co., 1920.

This book, which has appeared in a second enlarged edition, is a clear and concise account of a psychological method for the diagnosis of the intellectual level of children of the school age. The method adopted is largely based upon that employed by Binet and Simon, although certain modifications have been introduced as the result of experience gained from organized mental surveys in a number of American public schools. As an essential for statistical purposes the tests are standardized, but due recognition is given to the necessity for special records of variations in each individual examined.

The procedure here advocated, if used on a large scale by trained observers, should give invaluable information upon not only normal and abnormal juvenile mentality, but also the intellectual status of criminals and the feeble-minded.

Part 2 of the book is occupied with descriptions of the various tests and useful directions to examiners are appended.

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THE INNERVATION OF STRIPED MUSCLE-FIBRES AND LANGLEY'S RECEPTIVE SUBSTANCE.[1]

BY PROF. DR. J. BOEKE.

Utrecht.

Let me try to give you in this lecture in broad outlines the results of recent investigations on the mode of innervation of the striped muscle-fibres of vertebrates, the origin of the nerve-fibres which reach the muscle-fibres, their mode of ending on them, the manner in which the nerve-endings are connected with the muscle-fibres and the path by which the stimuli arriving by the nerves may reach the contractile substance of the muscle-fibre.

In the time at my disposal I must necessarily treat the subject somewhat dogmatically, and I fear that I shall not be able to do much more than express my own views about this subject without giving you a full historical account of the questions or stating the authorship of the large number of investigations carried out in this field and bearing on the innervation of the striped muscle-fibres of vertebrates and in general on the relation of the nerve-endings to their surroundings, on which these views are founded.

For a morphologist like myself it is only natural that morphological details will come in the first place to call our attention. But it seems to me that in general (apart from questions of phylogeny and comparative anatomy) morphological details derive their full value in the first place from the physiological and functional insight they are able to furnish us. For only when we are able to give a physiological interpretation of a given morphological detail can we say that we understand it, that it has given us some new insight into the ways of Nature.

But first let me state some general principles.

It cannot be denied that animal histology nowadays is in a period

[1] Lecture delivered at the University of London, on February 16, 1921.

of unrest, of evolution, of reform, that it is passing through a crisis, which is shaking its very foundations. The old conception of the cell as an elementary organism, established as the basis of all our researches after years of strife and an incredible amount of labour—this conception of the cell, on which generations of our teachers had built up the entire splendid structure of our knowledge of the animal body and its component parts, must be revised. And this process of revision has met with frequent and often serious opposition.

Obviously the cytologist, when studying and analysing the structure of the smallest elements that build up the living body, the cells and their derivations, is strongly tempted to regard these cells as units, as indivisible self-propagating independent elementary organisms, originated in a direct line from the dividing egg-cell and remaining independent units until their death or the death of the organism which they helped to build up.

This is not to be wondered at. It is not only that we have learned, since the time of Robert Brown, of Schleiden and Schwann, of Max Schultze, Huxley, Ranvier and Virchow, that all living bodies are composed of those small units, the cells, and their differentiation products. But afterwards there has been brought to light inside these cells themselves a distinct organization, a world in itself, a number of independent self-propagating structures, the nucleus, the amyloplasts, the tonoplast of de Vries and Went, the chlorophyll-bodies, the elaioplasts of Wakker, the centrosome, the mitochondriæ and their differentiation products, and in the nucleus the chromosomes with their outfit of chromomeres, which during the last years have become of such enormous significance, especially through the work of Morgan and his school on the physical basis of hereditary phenomena.

And we are here still standing on the threshold. Every new line of research started reveals to us unknown wonders of organization of the cells. But, on the other hand, when by the use of the more and more accurate and subtle methods of present-day histology, we penetrate farther and farther into that wonderful organization of the cells, the cell itself loses a part of its independence as an elementary organism. Many of them we see in the course of development losing their boundaries, and fusing together to form a syncytium. And even when, as seems to be the case in the myocardium of mammals, this syncytium in a later period of development is again differentiated into cells, even here the lines of junction of the cells are bridged across by the muscle-fibrils, and we are by no means sure

that the cellular elements, which we are able with tolerable accuracy to make out in the full-grown myocardium, are the selfsame elements which were seen to constitute the heart muscle tissue before the coalescing period.

The cross-striated voluntary muscle-fibres with their immense number of nuclei, formerly supposed generally to be formed by the elongation of a single enlarged cell, have now been shown to be the product of more than one cellular element.

In the first place they are built up by the joining together, end to end, of a number of cells of the muscle-plate, so as to form a syncytium, within which the striated fibrils make their appearance. But, secondly, even more than one segmental muscle-plate may contribute to the formation of a single muscle-fibre, as Godlewski and Sunier have shown and Agduhr was able to prove by their mode of innervation, and thirdly, as Sunier has shown, even mesenchymatous elements may take a part in the formation of the muscle-fibres.

As to the smooth muscle-cells, what we know about their development tends in the same direction. According to the observations of McGill the smooth muscle-cells of the alimentary canal of the pig, seemingly so distinctly separate as true cellular organisms in the adult animal, are developed from a syncytium of mesenchymatous cells surrounding the entoderm tube. Here too the myofibrils developing in their protoplasm need not be confined to the limits of a single-cell territorium, but may extend over two or even a number of cells. In the adult smooth muscle-cells the intercellular substance is bridged across by filaments passing from cell to cell, so that even the fully formed smooth muscle-tissue retains something of its syncytial character.

The same holds true for the follicle-cells of the ovary, for many forms of connective tissue, and, last and not least, for the elements of the nervous system. For, according to the observations of Held, during their development they seem to pass through a syncytial period that leaves them by no means the selfsame elements that went into it. And during their later development and in the course of the process of regeneration, as we shall see more clearly later on, the growing axons make use of conducting cells and other "mesenchymatous" or "nervous" elements to reach their destination.

In short, wherever we study the elements of the tissues more closely, and especially histogenetically, we get the impression that the cell-lineage of the cellular elements of the full-grown body is in

most cases by no means clear. Everywhere new elements are being formed, not always entirely independent of each other, and out of a syncytial stage these elements appear as new cells, independent of the old ones, which originally went into it.

Viewed in this light, the relation of the cell to the organism as a whole, to the individual, is changing too. The embryological experiments of recent years, for instance, the experiments in which two egg-cells were made to fuse together to form one giant individual of normal form and proportions, the brilliant experiments of Fischel, of Spemann and Lewis on the formation of the eye-lens out of different tissues, the experiments on regeneration and so many more splendid achievements of the most subtle latter-day operative technique, have placed this relation entirely on a different footing. We are everywhere struck with the exquisite harmony of the living organisms, a harmony between the different tissue elements, which demonstrates the domination of the individual over the elements which compose it, the cells.

And it is this harmony which alone can guarantee that equilibrium of the different parts of the organism which Nature is always and everywhere trying to establish or to re-establish when it was lost.

The conception of a struggle for life of the cells in the organism, worked out by Roux years ago to form the basis of his theory of functional adaptation as an analogon to the idea of a struggle for life in Nature, which Darwin revealed to us by turning upon it the full light of his genius, cannot be true, because it does not sufficiently take into account this harmony of the organism.

In Nature, the individual itself is fighting for its life and for the maintenance of its species. In this fight every individual is destroying remorselessly everything that is weaker, and only the fittest survive in the struggle. What we call the harmony of Nature is founded on our own artistic view of the things around us. In Nature itself it is only the elementary forces and energies that exist, which together build up that mighty symphony that the lover of Nature hears everywhere around him. When we speak of the unity in Nature, either we unconsciously transfer the idea of the individual organism to the conception of the universe, or we feel behind the forces and phenomena of Nature a Divine power, before which we can only bow our heads in profound submission, without trying to explain it.

But in the organism this harmony is a distinct reality. Every organism is a unity in itself, and the elements which compose it are

in the first place a part of the whole, deriving their full value not from themselves, but from the individual to which they belong. The individual, the organism as a whole, dominates the cells which compose it. It blends them together, so to speak, subjugates them, to form that unity of the living organisms, that harmony of their organization, which is the greatest wonder and mystery of Nature, and fills us with such a profound admiration.

It is not to be wondered at, that among the different parts of the organism it is the nervous system, which, if this conception is true, must in the first place exhibit phenomena in harmony with this view. And we do not look in vain.

It is not only that the neurone theory, the nerve-cell a distinct unity, even embryologically, histologically, functionally, a separate, independent organism, a true cell of the old school, has to be revised. It was shown by Held and his school, and afterwards I was able to confirm his statements in a series of admirable preparations by Dr. Heringa, that the fibres of the neuroblasts do not run free in the interstices between the cells, but that they everywhere follow distinct protoplasmatic paths either of migrant medullary elements or of mesenchymatous cells, until they reach their destination. In the adult animal too we never find free "naked" nerve-fibres running in the interstices between the cells of the tissues, not even inside the epithelium, as I hope to show you later on. Wherever we find a suitable object (as for instance the corpuscles of Grandry or of Meissner, the organ of Eimer in the mole, the muscle-spindles of the cross-striated muscles of different mammals, &c.) that enables us to study the course of the end-ramifications of the nerve-fibres, we can state definitely that nowhere does a neurofibrillar strand run free, independent of the surrounding elements. And in regeneration the same fact stands out clearly, amidst the often bewildering pictures we get in our preparations of the exuberance of the regenerating nerve-fibres.

Nor is this all. As I hope to show you in the course of this lecture, during the process of nerve regeneration after a nerve has been cut there exists, or is established, a perfect harmony between the elements of the different tissues which build up the path that has to be followed by the regenerating nerve-fibres. It is not only the nerve-fibre that is growing out and seeking its way to re-attain its original destination, but all the surrounding elements, the sheath-cells, the connective-tissue cells, the elements of the original end-

organs (either the sensory end-organs or the muscle-fibres) are seen taking a part in the regeneration process in perfectly harmonious co-operation, none remaining passive, none predominating in its action. And so the re-establishment of the nervous function in regeneration is not due to the simple outgrowth of the axon processes of the nerve-cells as independent units, but to the combining together of the forces of all the surrounding elements, to restore the harmonious equilibrium of the organism, which was disturbed by the degeneration of the peripheral end of the severed nerve.

But now to return to the subject of our lecture, the innervation of the striped muscle-fibres.

About the general structure of the motor end-organ of the higher vertebrates I need not enter into detail here. We know that the end branches of the myelinated motor nerve-fibres having reached

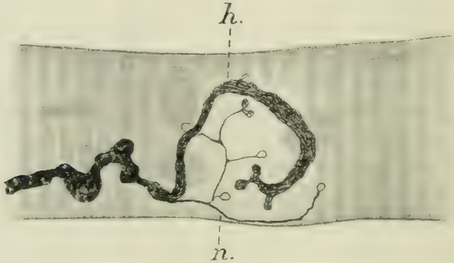

FIG. 1.—Normal motor endplate on a striped muscle-fibre of the hedgehog.

the muscle-fibres pass through the sarcolemma. The neurilemma of the nerve-fibre fuses with the sarcolemma and the axis-cylinder of the fibre is seen ending in a close terminal ramification with varicose expansions on its branches, which when appropriately fixed and stained show a fibrillar structure with reticulated or loop-like expansions. Having passed the sarcolemma, this ramification of the neuro-fibrillæ lies embedded in a layer of granular protoplasm (the sole), which is collected in a small mass at the place of the nerve-ending, and contains a number of nuclei.

Is this hypolemmal position of the nervous end-ramification only to be regarded, as Heidenhain contends, as an arrangement to attach the end-organ more strongly to the muscle-fibre ("Verankerung") and to prevent it from being torn from the muscle-fibre by a violent

contraction? I do not think so. There is no stronger mode of attachment of one element to another imaginable than that of the ends of the muscle-fibre to the tendon. And yet it is easy to demonstrate that, contrary to the well-known views of Oscar Schultze, the sarcolemma extends over the end of the fibre, the tendon-fibrils are attached to the outer surface of the sarcolemma (van Herwerden, Péterfi), and there is no actual continuity between contractile substance and tendon. Why then should such a continuity between the nerve-ending and the muscle-fibre be needed, only to prevent the nervous end-plate from being torn from the muscle-fibre by its contraction? In my opinion this hypolemmal position is only another example of the insufficiency of the old cellular conception, of the continuity of the different cellular structures with each other. And, as we will see further on, it is here of the utmost importance for the regular function of the motor end-organs.

FIG. 2.—Intraprotoplasmic ending o a nerve-fibre on a smooth muscle-fibre of the musculus ciliaris of the human eye.

In this connection it may be of interest to state, that in smooth muscle-cells the same peculiarity is met with. Here too the terminal nerve-fibrils pass into the cell, ending within the protoplasm with loop-like expansions, which often are seen lying so close to the nucleus of the muscle-cell, that they even make an indentation and thus are found lying in a shallow cavity in the side or on the top of the elongated nucleus, thereby proving their intraprotoplasmic position (fig. 2).

About the form of the neurofibrillar end-ramification I will say here a few words only. We know that the muscle-fibres of the different

classes of vertebrates possess characteristic forms of nerve-endings, from the curious spade-like expansion on the muscle-plates of amphioxus to the complicated ramifications of the mammalian motor nerve-endings. But this is not all. In every animal the end-ramification seems to represent a distinct type, so that the motor nerve-endings of a rabbit may be distinguished from those of a hedgehog or a bat; and, for instance, the nerve-endings on the muscle-fibres of a crow are of a coarser structure and form than those of the canary. Even in so

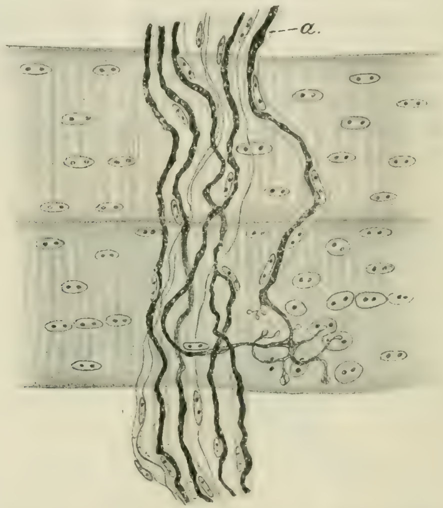

FIG. 3.—Regeneration of the motor endplates of the hedgehog, sixty-two days after the section of the nerves.

insignificant a detail as the form of the neurofibrillar network the nerve-ending appears to be in exquisite harmony with its surroundings, a part of the whole and not a thing in itself. A striking example of this harmony is again furnished by the regeneration process. When a motor nerve has been severed, after a certain lapse of time functional continuity between the cut ends may become re-established by an outgrowth of new fibres from the central stump, the newly sprouting fibres finding their way through the scar tissue between the cut ends of the nerve. After having traversed this tissue, they pass towards the periphery along the course of the degenerated fibres, the sheaths of

which serve as guides for the down-growing axons, until they reach their destination, the muscle-fibres deprived of their innervation by the cutting of the nerve. Then there are formed on those muscle-fibres end-ramifications (fig. 3) ; these show an exuberance of delicate nerve-fibrillæ, growing out between and on the muscle-fibres, forming end-organs of the most bizarre and complicated structure, entirely different from the normal motor-endings, of which they are the substitutes, especially in their exuberance of form and abundance of ramifications. In this way functional continuity is re-established, nerve-impulses are able to reach the muscle-fibres, contractility and voluntary movement is restored.

But the process of regeneration is not finished yet. When we keep the animal alive some months after functional activity is restored, we find the regenerated motor nerve-endings again changing their form. The complicated, aberrant end-plates, with their exuberance of branches, so totally unlike the normal end-plates, are gradually disappearing. Superfluous expansions of the neurofibrillar structures seem to be drawn in. New smaller branches are formed, and by degrees we see, in the course of a few months, everywhere reappear the normal form of the nerve-endings, characteristic for the species studied, until the equilibrium of form between the different elements constituting the muscle is restored. Only then does the regeneration process come to an end. It is this equilibrium of form, this perfect harmony between the elements, that Nature aims at, and not only the restoration of function.[1]

To this end, as I have mentioned before, all the elements of the surrounding tissues are acting and striving together. It is not only the nerve-fibre that is growing out and trying to reach its original destination. The scar tissue developing between the cut ends of the nerve, especially in the beginning of the regenerative phase, is built up of delicate, elongated, branched cells, fused to form a syncytium ; about these cells we are not sure whether they are entirely of nervous origin (cells of the nerve-sheaths, lemmoblasts), or derived from the connective tissue, of mesenchymatous origin. These syncytial cells are found to enclose the growing axons, direct them towards the peripheral end of the nerve, and so form a conducting tissue for the outgrowing fibres. The delicate neurofibrillar strands are, as far as can be made out, always

[1] For further details in these matters I may refer the reader to the article : "Nervenregeneration und anverwandte Innervationsprobleme," in the *Ergebnisse der Physiologie*, xix Jahrgang, 1921, where the literature bearing on the subject is to be found.

found inside these elements, running intraprotoplasmically from one cell of the syncytium to the other, until they reach the peripheral nerve-end. Here again a syncytium is formed, the cells of the neurilemmal sheath of the degenerated nerve-fibres having proliferated and fused together into long protoplasmic strands occupying the old nerve-sheaths. The regenerating outgrowing nerve-fibres are drawn towards these protoplasmic strands or conducted to them by the elements of the scar tissue ("hodogenesis" of Dustin), and enter them, here again running intraplasmically. When the regenerating nerve-fibres have traversed the peripheral nerve-end, and have reached their destination, they again are drawn towards the muscle-fibres. We must suppose that the sarcoplasmic soles of the old degenerated endings exercise a neurotropic and neurocladic influence upon the ingrowing nerve-fibres,

FIG. 4.—Formation of collateral outgrowths of the sheath-cells of a regenerating motor nerve-fibre on a muscle-fibre of the hedgehog.

owing to which they are drawn towards them, enter them and immediately form a number of branches. These branches, collateral and terminal, form together the new nerve-endings described above. It is obvious that the most valuable phase of the regeneration process is the formation of these new end-organs, for without them no functional continuity ever can be obtained. And it is wonderful to see how all the forces of the organism are called into play here, acting together in harmonious co-operation to restore the lost equilibrium. It is not only that the outgrowing nerve-fibres are conducted towards their destination by the protoplasmic strands of the old nerve-sheaths, and that the sarcoplasmic soles attract them and cause them to form their terminal ramifications on the muscle-fibres. First, we see the cells of the nerve-sheath of the nervous plexus between the muscle-fibres beginning to proliferate, to form collateral protuber-

ances, outgrowths, reaching the muscle-fibres (fig. 4), establishing new connections into which and through which the outgrowing nerve-fibres pass to their destination. Secondly, the connective-tissue elements appear to build up a new syncytial conducting tissue throughout the atrophied muscle to the same purpose, the formation of new ways for the outgrowing nerve-fibres. It is true that this last-named proliferation of the connective-tissue elements is by no means easy to follow with sufficient clearness in the sections through the close-packed muscle-fibres of the common muscles. But let us examine places where we find the muscle-fibres more loosely arranged, as is the case, for instance, in the muscle-spindles. Here, as it was described so well by Sherrington, we find the axial spindle-fibres completely surrounded by a large lymph space, bridged across and partially subdivided in

FIG. 5.—Conducting tissue of the periaxial space of a muscle-spindle of the hedgehog, with neurofibrillar strands.

many points by extremely tenuous membranes, filaments, and strands of syncytial connective-tissue elements. And here we can state definitely, not only that the end-ramifications of the normal nerve-fibres are everywhere enclosed within the protoplasm of these mesenchymatous cells (fig. 5), but that during the degeneration phase of the regeneration process these syncytial cells proliferate, branch and form numerous new connections, so that a typical conducting syncytium is formed which catches up the ingrowing fibres, encloses them and enables them to reach their destination, the axial muscle-fibres.

And now, after having studied these things in the muscle-spindles, when turning again towards the ordinary muscle-fibres, we detect everywhere traces of the same process going on here, of the same conductive tissue forming between the muscle-fibres. And I can imagine no other

process which affords us such a striking example of the harmonious co-operation of the elements of the different tissues as subordinate parts of the whole, to reach a given end, the restoration of the equilibrium of the organism, than this mode of regeneration of the motor nerve-endings.

The most prominent feature of the motor nerve-endings of vertebrates (and, as far as they have been studied, the same holds true for those of invertebrates) is the expansion of the neurofibrillar structure. As soon as the axis-cylinder passes through the sarcolemma we see the bundle of neurofibrillæ, after a momentary constriction, broaden out, disperse and expand into branched networks or looplike structures. A typical example of this expansion is exhibited in the figures. Now it is well known that ever since these neurofibrillæ were described by Apathy, who succeeded in staining them in his preparations with the most wonderful accuracy, not only their arrangement, connections and other morphological details have been under constant discussion, but morphologists and physiologists alike have been discussing the question of the function which we have to ascribe to these peculiar fibrils, which are found without exception in all parts of the ganglion-cells and their processes. Apathy himself regards them as the sole conductors of the nerve-impulses, and, in the lecture delivered at Cambridge in 1898, gave a number of arguments in support of this conclusion. In later years it was chiefly Bethe who took up the idea and carried out several interesting experiments to furnish proofs for it. A number of investigators agreed with him. Others, such as Wolff, von Lenhossek, Verworn, Jenkins and Carlson, Cajal, are convinced that the nervous impulses are also conducted by the neuroplasmic substance enveloping the neurofibrillar structure, either by the two substances together or by the neuroplasm alone. In this case the neurofibrillæ only play the part of supporting filaments of the nervous elements. It would lead us too far to review all the different statements and arguments of the numerous authors who have contributed to the question under discussion. In my opinion there is only one conclusion to be drawn from the different observations on this point, and that is, that we have to see in the neurofibrillæ the actual conductors of the nerve-impulses.

But if this assumption be true, we are confronted with a real difficulty. In general there seems to be no connection between the neurofibrillar structure of the motor-endings and the contractile substance of the muscle-fibre. Even Kühne, who advocated so strongly the hypolemmal position of the motor-endings, pointed out (in the Croonian

Lecture) that properly stained hypolemmal branches terminate usually in rounded ends sharply marked off from the muscle substance. According to Langley, even in the simplest case, that of the nerve-endings of amphibia, where the ramifications spread over a considerable length of the muscle-fibre, the substance which has been described as continuous with the endings is clearly the sarcoplasm of the muscle, which stretches throughout the muscle-fibre, and not the contractile substance itself. And indeed, as far as my experience goes, I know of only one case in which the neurofibrillar structure (or something akin to it) is found to be continuous with the myofibrillæ. That is in amphioxus, where I found the extremely delicate terminal meshes of the neurofibrillar network to be continuous with the anisotropic discs of the muscle-plates, each anisotropic disc receiving a small branch

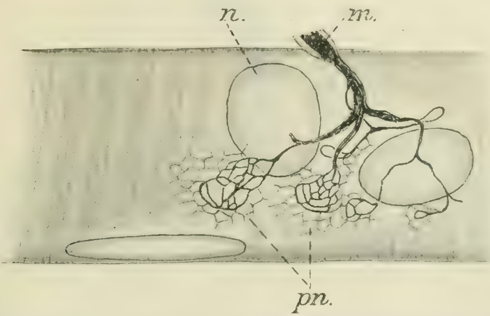

Fig. 6.—Normal motor endplate of the bat, with periterminal network; *pn.* = periterminal network; *n.* = nucleus of the sole.

from the neurofibrillar structure running along it. In all the other vertebrates there lies, when studied in ordinary preparations, a stretch of undifferentiated granular sarcoplasm between the terminal ramifications of the neurofibrillæ and the contractile substance, the myofibrillæ.

But let us study these things in carefully stained and differentiated Bielschowsky preparations, in which not only the neurofibrillar network stands out very clearly, but the adjoining tissues and elements have taken the stain, too, so as to be visible in the sections. Then there appears in the sarcoplasm surrounding the neurofibrillar end-ramifications a reticular differentiation, extremely delicate, on one side attached to the neurofibrillar ramifications, on the other side losing itself in the sarcoplasm. In my first publication I proposed for it the name of

"periterminal network," and by this name I will call it in the following pages. Enclosed in the finely granular sarcoplasm we find this periterminal network as extremely delicate fibrils, forming a reticulum with small meshes and somewhat thickened knots, which is continuous with the neurofibrillar ramifications (figs. 6, 7). Especially around the end-loops and end-nets of this structure the meshes of the periterminal network are very distinct; they appear here either as small regular, polygonal meshes of about the same size, or those surrounding somewhat larger end-loops or end-reticula of the neurofibrillar structure are of somewhat larger dimensions, slightly elongated, crescent-shaped or cup-like in appearance. But even where the innermost meshes, surrounding

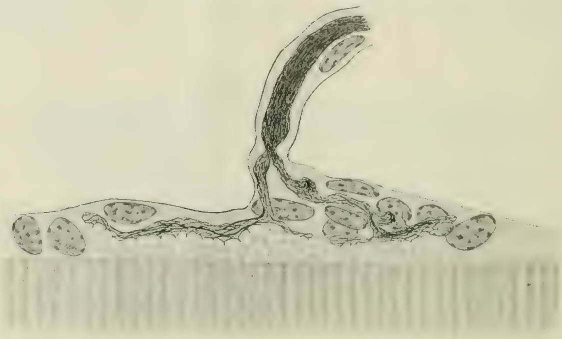

FIG. 7.—Normal motor endplate on a muscle-fibre of the m. obliquus sup. of the cat, with periterminal network, seen "in profile."

the end-loops of the neurofibrillar structure, appear in this way, the second layer of meshes generally shows the polygonal, regular form, mentioned above. There where the reticulate neurofibrillar end-loops are not spread out in one plane, but are three-dimensional, more or less pear-shaped, the meshes of the periterminal network are seen surrounding them on all sides.

But it is not only with the end-loops and end-reticula of the neurofibrillar structure that the periterminal network is found to be in connection; it may be seen as clearly surrounding the more or less varicose branches of the motor-ending, and here too it is always found in close connection with the neurofibrillar structure (fig. 7). Often, when examined under the highest power, the neurofibrillar strands give the impression

of being covered with extremely tenuous spines. With these tiny spines the fibrils of the periterminal network are seen to be in connection. The meshes of the periterminal network appear to be continuous throughout the sarcoplasm of the sole, and although it is wont to take a stronger stain in the immediate neighbourhood of the axial neurofibrillar ramification, in several cases I could follow the fibrils of the periterminal network throughout the sarcoplasmic sole to the myofibrillæ; here they seem to pass into the anisotropic discs of the myofibrillæ, and to form an extremely delicate network throughout the whole thickness of the discs (fig. 7).

As to the morphological value of the periterminal network I need not enter into details here. The time at my disposal prevents me from going too far into historical details. Suffice it to say that traces of this network seem to have been seèn by Retzius, Koelliker and Rollett, that it makes the impression of being an element *sui generis* and that in my opinion it is not identical with the reticular differentiations inside the muscle-fibre described by Veratti, Cajal-Fusari and Holmgren.

The periterminal network is an element *sui generis.* But is it to be regarded as of a neural nature, a continuation of the neurofibrillar structure, or is it a differentiation of the sarcoplasm itself? This is a difficult question to answer. It is always found in close connection with the neurofibrillar structure. During the development of the motor end-plates it is always found to appear as a continuation of the neurofibrillar structure. Even in properly stained sections it always takes the darkest stain where it is attached to this structure, and it is often only visible in this part of the sarcoplasm and gradually disappears from view farther on. And yet its features are so different from the neurofibrillar structure, and it gives so clearly the impression of a protoplasmic reticulum, that I do not hesitate to assert that in my opinion it is of protoplasmic (sarcoplasmic) origin, but that it develops only in connection with and under the influence of the neurofibrillar structure of the motor nerve-ending. This is important for the accurate conception of its physiological and functional significance.

An interesting point is its behaviour during degeneration and regeneration of the motor nerves. The most characteristic feature of the degeneration of the motor nerve-endings, after cutting of the nerve, is the gradual disappearance of the neurofibrillar network. Within a few hours after the operation the fibrils begin to swell, to fuse together, and the mass thus formed, in the course of one or two

days breaks up into portions and disappears. During the first stages of the process of degeneration the periterminal network may still be distinguished, but it disappears from view together with the neurofibrils, and only the granular sarcoplasm of the sole remains visible. When, after a lapse of time, the regenerated motor nerves again reach the muscle-fibres, their ends ("cônes de croissance") pierce the sarcolemma, come again in contact with the sarcoplasm and develop into new nerve-endings. As soon as the muscle-fibre is reached and the first neurofibrillar loops have entered it, the periterminal network makes its reappearance (as is shown in fig. 8) as a reticular

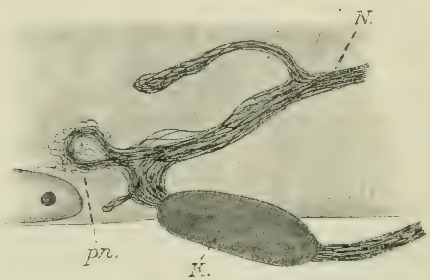

FIG. 8.—Regeneration of a motor endplate of the hedgehog, with formation of the periterminal network (*pn.*); *K.* = nucleus.

differentiation of the sarcoplasm surrounding the neurofibrils, at first only in the immediate neighbourhood of the neurofibrillar strands, but gradually spreading and growing out. So its disappearance and its reappearance are absolutely connected with and dependent on the presence or absence of the neurofibrillar structure.

All this seems to indicate that the periterminal network (whether as a sarcoplasmic reticulum or a continuation of the nervous structure) is intimately connected with the functions of the neurofibrillar structure of the motor-endings. And thus, when we have every reason, as I have mentioned already, to regard the neurofibrillæ as the actual conductors of the nerve-impulses, the assumption seems to be justified that the neurofibrillar structure, not being continuous with the contracting substance of the muscle-fibre, the impulse is carried on by the periterminal network and in this way transmitted to the contracting substance of the muscle-fibre, the myofibrillæ.

This may be brought into connection with some well-known physiological facts. In his brilliant Croonian Lecture for 1906, Langley has shown that there is every reason to suppose that in the case of a connection of the peripheral end of an efferent nerve with a cell there must be present in the cell one or more substances (which Langley calls "receptive substances") capable of receiving and transmitting stimuli and capable of isolated paralysis. According to Langley it is this substance which is stimulated or paralysed by poisons ordinarily taken as stimulating or paralysing nerve-endings (curari, nicotine, strychnine, and others). Probably not only the function of reacting to numerous chemical bodies, but also the special liability of both afferent and efferent nerves to fatigue, must be transferred from the nerve-endings to the same constituent of the cell. The nerve-ending itself is, according to Langley, physiologically not essentially different from the nerve-fibre. Therefore the special action of the poisons mentioned above must be due to the presence of one or more receptive substances in the cell. These substances are, Langley considers, radicles of the protoplasmic molecule, which reminds us of the haptophoric groups of the "Seitenketten" theory of Ehrlich; and indeed, I think that the general opinion amongst physiologists is, that a morphological substratum of the receptive substances (or the "neuroplasmatische Zwischensubstanz" of Asher) is not to be looked for in histological preparations. And not only physiologists. Even in morphological work, as for example the study of Sutton on the development of the neuro-muscular spindle in the extrinsic eye-muscles of the pig (1915), the conclusion is drawn, that until now there does not exist a morphological basis for the receptive substance.

And yet the later work of Langley and the work of Keith Lucas on the different behaviour of the neural region of the muscle-fibre, and the aneural region as regards the action of poisons, lend support to the view that such a substance exists in proximity to the nerve-endings in muscle. And it was shown by Noel Paton and Findlay in 1916 that after the efferent nerves have been cut and the nerve-endings have degenerated, the receptive substance in the muscle-fibre seems to degenerate secondarily to the neural endings. It seems to remain unaltered for some time after the nerve has ceased to act, and in some cases have its excitability modified during the process of degeneration of the nerve; after some time, however, it degenerates also.

Now all this seems to me to point to the conclusion that we may look for a well-defined, sharply localized morphological basis for the

receptive substance, that it has to be looked for inside the sarcoplasm, between the nerve-ending and the contractile substance, and that all the conditions for it are satisfactorily fulfilled by the "periterminal network" described above. It lies embedded in the sarcoplasm and indeed seems to be a part of it, it is in close connection with the neurofibrillar structure of the nerve-ending and is especially visible in the immediate neighbourhood of it, but extends to the contractile substance. It has a morphological aspect different from that of the neuro-fibrillar structure of the nerve-ending, and seems to be of a finer texture in highly developed active muscle-fibres (as the extrinsic eye-muscles of the cat for instance) than in the muscle-fibres of slower action (as the muscles of the tongue of the hedgehog). And last not least, it degenerates secondarily to the degeneration of the motor-endings, and again makes its appearance as soon as the regenerating nerve-fibres have reached the muscle-fibres and are forming new end-ramifications in the sarcoplasma.

But, as a physiological conception, the receptive substances of Langley are not confined to the striated muscle-fibres. And indeed, one is tempted to ask whether in every case of a synapse, at least of a connection of a nervous end-ramification with a peripheral element, either of an efferent or an afferent nature, a receptive substance, capable of receiving or transmitting stimuli, has not to be localized as an intervening medium between the undifferentiated protoplasm and the neurofibrillar structure of the nerve-ending.

There are several histological phenomena that seem to point in this direction. In the first place I have to mention the connections of the sympathetic nerves with the striated muscle-fibres. Some years ago it was shown that besides the endings of the spinal motor nerves on the muscle-fibres each fibre receives a branch of bundles of fine non-medullated nerve-fibres running between the muscle-fibres. These fibres clearly belong to the automatic nerve-system. For it could be shown that when the eye-muscle nerves (for instance the trochlearis nerve) are sectioned as near as possible to their origin from the mid-brain, before they receive a branch from the autonomic nerve-plexus, the motor nerves and their endings and the sensory nerves degenerate and disappear, but the non-medullated sympathetic nerve-fibres and their endings on the muscle-fibres remain unaltered. Later on it was shown by Dusser de Barennes and Boeke for the trunk muscles, and by Agduhr for the muscles of the extremities, that, when the spinal roots are cut and the spinal ganglia removed without injuring the sympathetic roots (ramus communicans albus et griseus), the common motor-

endings and the sensory endings disappear, but the fine non-medullated nerve-fibres with their small nerve-endings on the muscle-fibres remain unaltered. The sympathetic fibres for the muscles of the tongue seem to run, not in the hypoglossal, but in the chorda tympani (Boeke). Their endings on the muscle-fibres present the same aspect as the sympathetic endings in the muscles mentioned above. So the sympathetic innervation of the striated muscle-fibres seems to be put on a solid morphological basis.

Now these sympathetic nerve-endings have the same hypolemmal position as the common motor-endings, and indeed are often found embedded in the same mass of granular sarcoplasma. Moreover it is certainly of interest that surrounding the end-loops and end-nets of these sympathetic nerve-endings there may be distinguished exactly the same regular protoplasmic network as was described for the motor-endings. And indeed, where these sympathetic endings were found embedded in the sarcoplasm of the motor-ending itself, the same periterminal network was seen surrounding them both.

For smooth muscle-cells too, as I mentioned above, it was not only possible to show that the end-loops of the nerve-fibres enter the cell and lie embedded in the protoplasm, but here too they seemed to be in connection with a distinct reticulum.

As to the sensory nerve-endings, there are at least two different kinds of tactile end-organs which exhibit the same features. In the first place the corpuscles of Grandry in the skin covering the duck's bill (figs. 9, 10). As is known, these corpuscles consist of two (or three) large, flat, disc-like cells within a capsule, with the axis-cylinder terminating in a flattened expansion between the cells. In the numerous papers dealing with the histology of the corpuscles of Grandry the tactile disc is always regarded as being entirely independent of the two cells between which it is lying. But Gasiorowsky saw these cells degenerate after section of the nerve, and in a set of admirable Bielschowsky preparations of the skin of the duck's bill made by Dr. Heringa in my laboratory, not only the tactile disc could be seen in a syncytial connection with both cells, but inside the protoplasm of these cells there was visible a distinctly stained, sharply defined network in absolute continuous connection with the neurofibrillar structure of the tactile disc. It is seen extending throughout the whole thickness of the two adjoining cells, in short, it exhibits all the features so characteristic of the periterminal network surrounding the motor nerve-endings.

In the second place I have to mention the tactile discs in the epi-

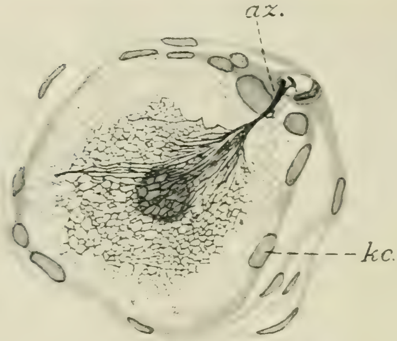

FIG. 9.—Tangential section of a corpuscle of Grandry of the skin of the duck's bill, with intraprotoplasmic network.

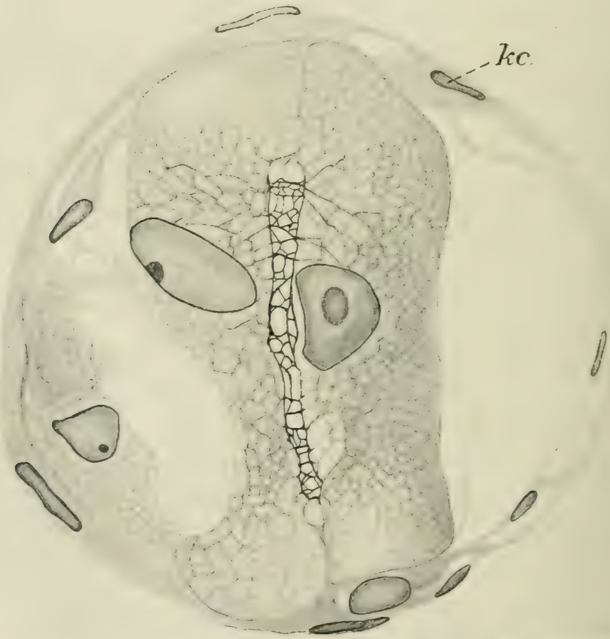

FIG. 10.—Cross-section of a corpuscle of Grandry of the skin of the duck's bill, with intraprotoplasmic network; *kc.* = capsule-cells.

dermis. It is generally assumed that these curious flattened or crescentic nervous expansions lie in the interstices of the deeper epithelium cells, for example, of the pig's snout, or in the organ of

FIG. 11.—Cross-section of the epithelium of the organ of Eimer of a mole, with nerve-fibres entering the skin and a tactile cell with nerve-fibre and intraprotoplasmic network (*pn.*).

FIG. 12.—Transverse section through the epithelium of the skin of the pig's snout with two tactile cells of Merkel, showing the intraprotoplasmic position of the neuro-fibrillar network.

Eimer in the snout of the mole, and they may be shown to consist of a fine network of neurofibrillæ. From the earlier investigations of Merkel and Ranvier to recent times almost all writers on the subject agree on

this point, that they are lying between the cells of the epidermis. And yet, when we study them in very thin sections treated, fixed and stained with the utmost care, we come to the conclusion that they are not lying between the cells with a somewhat differentiated cell of the epithelium covering them, but that the neurofibrillar expansion, the tactile disc, lies inside the protoplasm of a differentiated cell of the epidermis. And, moreover, we see that the neurofibrillar reticulum is continued into a distinct protoplasmic network with fine regular meshes, extending throughout the cell (fig. 11) and exhibiting just the same features, characteristic of the periterminal network, as in the case of the corpuscles of Grandry.

These points, which can only be seen in the most carefully made preparations and with the use of the highest magnifying power, are not at all easy to study, and although I was able to make several observations on other sensory endings, as, for instance, the corpuscles of Herbst and the cells of the taste-buds, which seem to tend in the same direction, I do not think it advisable to give here the further details of these investigations.

But it seems to me that the observations recorded give us reason for supposing that not only in the case of the motor-endings but also in the different forms of the sensory nerve-endings there exists in the first place a close connection between the nerve-endings and the surrounding "lemmoblastic" elements that gives us a right to speak of a syncytial arrangement. And, secondly, we may assume that everywhere between the neurofibrillæ of these nerve-endings and the protoplasm is inserted a distinct reticulum, which seems to be the histological substratum for the receptive substance of the physiologists.

This may furnish a solid base for further study in this direction, and for the present-day histologist it is another proof of the inadequacy of the old cell theory, which regards the cells as independent self-sustaining units, and of the wonderful harmony in which all those units combine and act together to build up and sustain the higher organism, the individual.

THE RELATION OF THE HIND-BRAIN TO MICTURITION.[1]

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In a previous paper [1] experiments on the cat were described, from which, among others, the following conclusions were drawn :—

(1) There is a motor tone passing to the bladder through the pelvic nerves which arises in the central nervous system above the level of the lower thoracic region of the cord.

(2) Transection of the cord in the lower thoracic region abolishes reflex micturition, i.e., the reaction which ultimately occurs if a bladder is gradually artificially distended, leading to the expulsion of its contents.

(3) Transection of the cord in the lower thoracic region permanently abolishes micturition. The escape of urine which occurs when the stage of so-called spontaneous (or automatic) micturition has been reached differs from micturition in the essential respect that the bladder contraction and urethral relaxation are not co-ordinated, as is shown by the fact that the bladder is never emptied. When this escape of urine is provoked reflexly by perineal stimuli, the jets of urine passed can be seen to be synchronous with rhythmic contractions and relaxations of the perineal muscles.

The object of the present investigation was firstly to determine the level in the central nervous system of the origin of the motor tone of the bladder muscle. Secondly, to find out what parts of the central could be removed and micturition still take place. Thirdly, to find what simple reflexes are combined together to form micturition.

(1) Motor Tone of the Bladder Muscle.

The state of the bladder muscle was estimated, as in the previous experiments, by measuring its volume under a constant pressure of 10 cm. of water. The method, which was described before, consisted in cutting away the pubic arch, tying a brass catheter (size 10 Charrière)

[1] The expenses of this research were defrayed by the Graham Research Fund.

into the urethra just above the prostate in the male, and in the corresponding place in the female, and allowing all urine to escape from the bladder. The catheter was then connected with a closed graduated wash-bottle of water hung on pulleys in a bath which was kept warm and at a constant temperature within 3° C. The bottle was raised or lowered as the volume altered, except for the rhythmic undulations of contraction and relaxation which are usually present: 30 c.c. in the bottle caused a rise of pressure of just under 1 cm., hence as the undulations seldom produce a volume difference of more than 15 c.c., the maximum pressure variation would usually be about ½ cm. of water. The volume was recorded by connecting the air space of the bottle with a Langley's piston recorder which was attached to a Keith Lucas' lever. As previously stated, the volume of urine secreted by cats under anæsthetics is negligible for the purposes of these experiments.

FIG. 1.—Cat, female. Ether. Splanchnic nerves divided. Bladder volume at 10 cm. H_2O pressure and 42° to 43° C. At the signal the spinal cord was divided between the first and second cervical roots. Time marker, five seconds.

A series of experiments was first made to find out if the same effect on the bladder volume was produced by dividing the cord in the upper cervical region as has been previously described in the case of the lower thoracic region [1], namely, an immediate abolition of any sharp undulatory contractions and a dilatation which persists, the dilatation being sometimes preceded by a sharp transient contraction. For this purpose the cats were anæsthetized with ether, a tracheotomy tube with two exits tied in the trachea, the catheter tied in the urethra as described, and dorsal part of the arch of the atlas cut away and the dura deep to it opened. Artificial respiration was then started and ether given with the respiration pump. After the volume had become reasonably constant, the cord was divided and the result on the volume noted. The effect was apparently exactly the same as in lower thoracic

transections and occurred with remarkable constancy (fig. 1); the only cases in which it appears not likely to occur are those with very large volumes and no undulatory contractions, a condition which is generally due to very deep anæsthesia or a bad general condition as a result of hæmorrhage. As in lower thoracic transections, division of the cord in the region of the first cervical root is followed by a persistent increase in volume and abolition of sharp rhythmic contractions, whether the hypogastric nerves are intact or divided. There are two factors in the upper cervical transection which might affect the bladder volume that do not occur in the lower thoracic region, namely, the fall of blood-pressure and the alteration in the amount of ether. That the effect is not due to the fall of blood-pressure was shown by experiments in which the cord was divided after previous division of the splanchnic nerves, the effect on the bladder volume being the same as when these nerves were intact. When the cats were having artificial respiration, as a rule, spontaneous breathing neither ceased nor was synchronous with the pump; they were, therefore, getting less ether before the spinal transection than after, when spontaneous respiration ceased, the ether regulator not having been altered: no steps were taken to find out what this difference was and, as will be shown later, ether both abolishes rhythmic undulations and causes a relaxation of the bladder in decerebrate cats: in this case, however, there is a very appreciable though not long interval before the effect occurs, whereas in the transection it is immediate (figs. 1 and 4, Part I), from which it seems that the effect cannot be due to any increase in the amount of ether taken after spontaneous respiration ceases. It was therefore concluded that the motor tone of the bladder arises in the brain.

A series of experiments was then made to determine whether the same effect on the bladder volume was produced by division of the mid-brain as by division of the cord. For this purpose the cats were tracheotomized and ether continued with an artificial respiration pump. A large part of the skull in front of the tentorium was removed and the dura opened. The brain section was made with a blunt knife, keeping it close against the tentorium. In some cases the brain in front of the section was removed in order to allow all hæmorrhage to come forwards and thereby avoid an increase of pressure in the posterior fossa. Two of the experiments were useless as death occurred a few minutes after the brain section. In the remaining five, one showed an abolition of undulatory contractions and a persistent increase in volume as is the case after division of the cord, one (fig. 2) showed an increase in volume

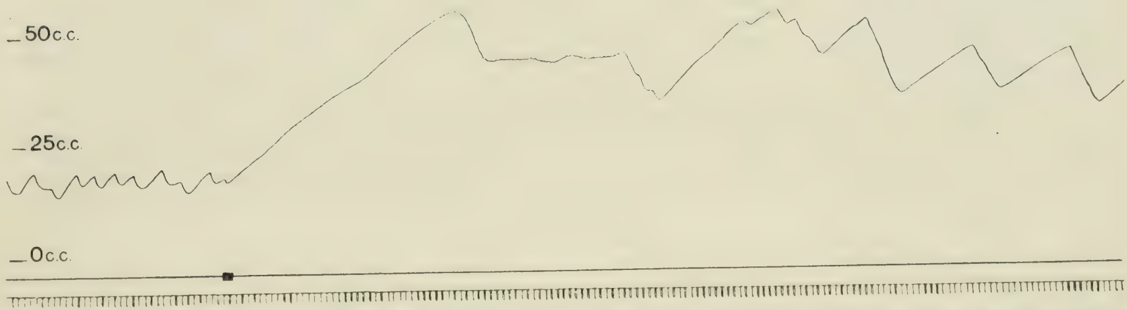

FIG. 2.—Cat, male. Ether. Bladder volume at 10 cm. H_2O pressure and 39° to 41° C. At the signal the mid-brain was divided through the superior colliculi and crura just in front of the superficial origins of the third nerves. Time marker, five seconds.

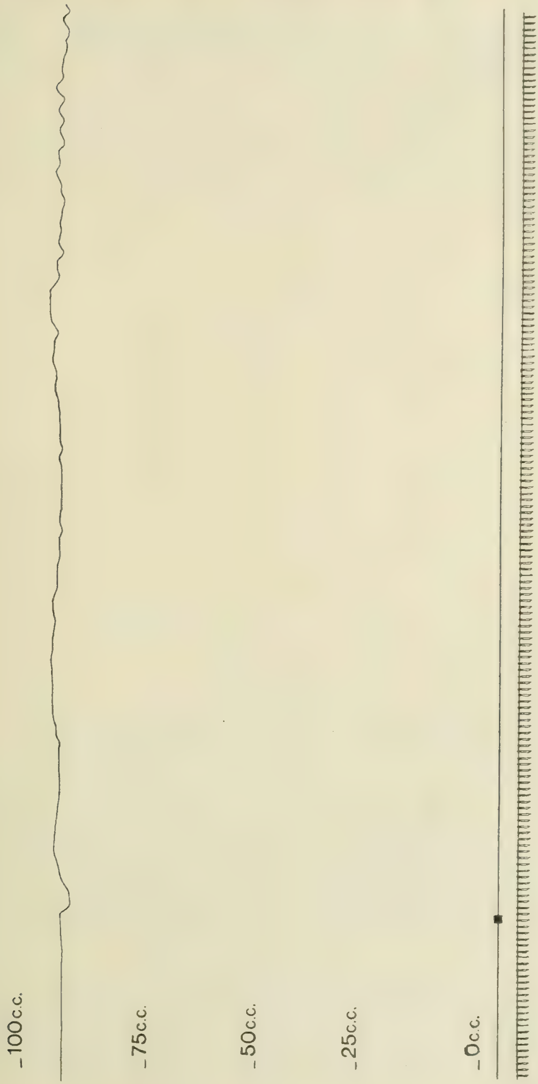

FIG. 3, Pt. I.

but large undulations became established again and the remaining three all showed diminution of volume with large undulations: in one of them (fig. 3, Parts I and II) the diminution took some time to begin, in the other two it was immediate. In the three experiments where a contraction followed the division of the mid-brain subsequent division of the upper cervical cord or lower part of the medulla was followed by the usual effects (fig. 3, Part III), namely, a persistent increase in volume and abolition of all large undulatory

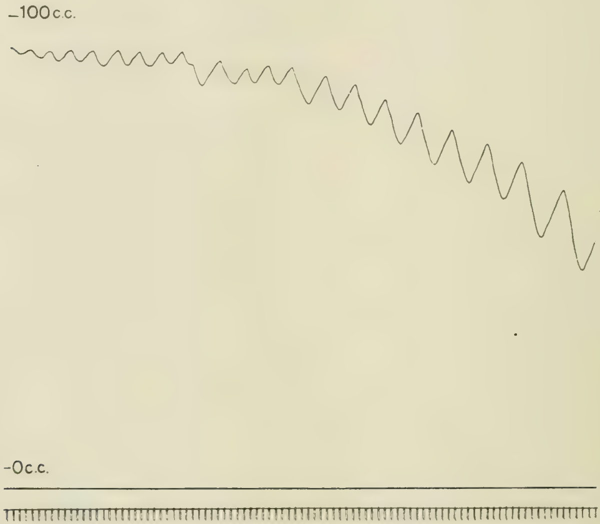

Fig. 3, Pt. II.

contractions. From these experiments it was concluded that the motor tone of the bladder arose somewhere between the lower part of the mid-brain and the lower part of the medulla. Subsequent experiments to localize its level of origin were made on decerebrate cats without anæsthetics.

Decerebration was performed under chloroform anæsthesia. The origin of the temporal muscle was detached from the skull. A trephine hole was made just in front of the position of the tentorium on the left side: this was enlarged with bone forceps and a large hole made in the dura. The division of the mid-brain was made as already stated and

the whole brain in front of it rapidly removed with the handle of a teaspoon. As stated by Bazett [2] the bleeding is very much less if the anæsthesia is very deep. The anæsthetic was stopped directly the the brain was removed. Bleeding was stopped by plugging the skull;

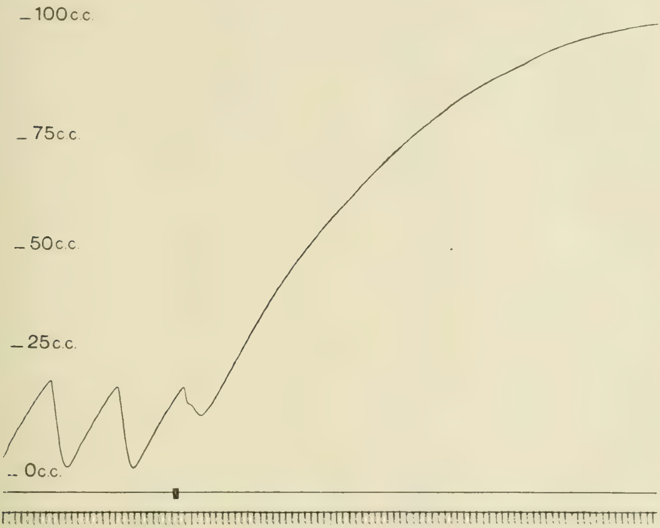

FIG. 3, Pt. III.

Cat. Female. Ether. Bladder volume at 10 cm. H_2O. At the signal in Part I the brain was divided just in front of the superior colliculi and the stalk of the pituitary.

Part II is ten seconds after Part I.

Part III is about six minutes after Part II. At the signal the cord was divided between the first and second cervical roots. The volume remained approximately the same till the animal was killed twenty minutes later. Time marked, five seconds.

In Parts I and III there was the same amount of air in the reservoir and piston recorder; the volumes represented in these two therefore correspond accurately. In Part II it was thought necessary to alter the amount of air in the reservoir to prevent the needle going off the tracing: after the experiment the 100 c.c. mark on this was made to correspond to that on Parts I and III: the other volumes of Part II do not, however, necessarily correspond accurately to those of Parts I and III, as with a Keith Lucas' lever a given change of volume does not produce the same excursion of the needle in all positions.

Horsley's method with a piece of freshly cut muscle placed over the sella turcica and plugged down with wool was found very effective. More than half an hour was always allowed to elapse before proceeding with the experiment. The volumes of decerebrate cats' bladders at

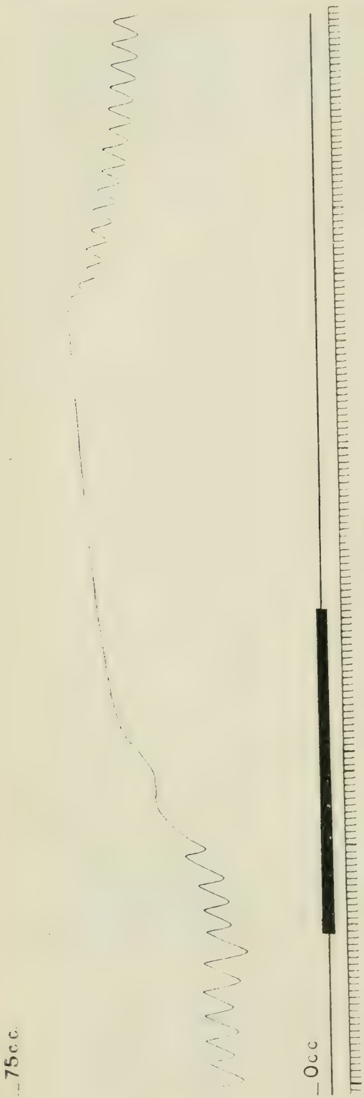

Fig. 4, Pt. I.

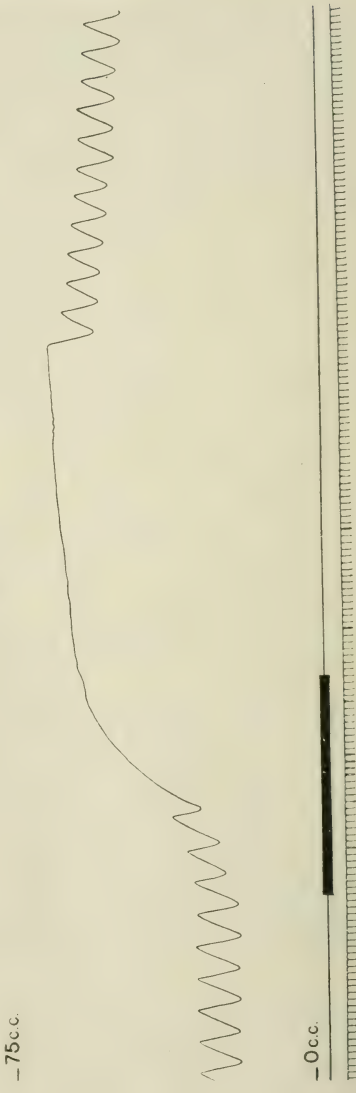

Fig. 4, Pt. II.

10 cm. of water pressure were on the whole much lower than those of intact cats under ether.

Before proceeding further with experiments to determine the level in the brain at which the motor tone of the bladder arises it was thought advisable to find out the effect of curare and also of ether and chloroform on the bladder volume of decerebrate cats. Chloroform and ether were given by putting one exit of a three-way tracheotomy tube into the air space of a Wolff's bottle containing some of the corresponding liquid, the second exit being clamped to a greater or lesser degree: by this means rather concentrated vapour was given. In the three experiments which were made the effect was the same: the volume gradually increased, in about a minute the undulatory contractions ceased and the

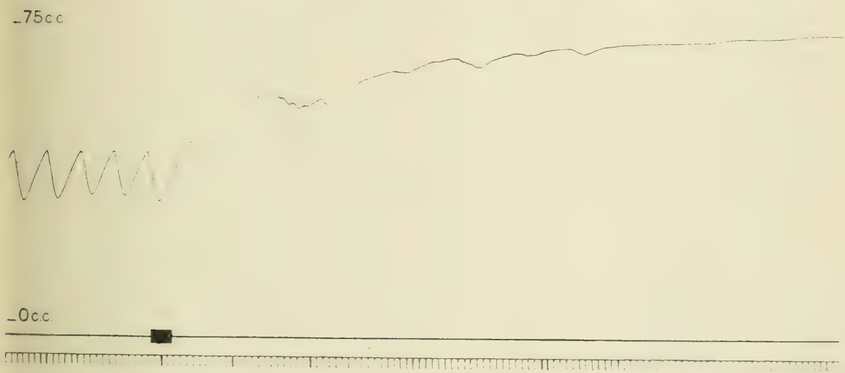

FIG. 4, Pt. III.

volume then increased more rapidly (fig. 4). It is the same effect as that of dividing the cord but it is not produced as suddenly. To show the effects of curare on the bladder volume, a solution of it, made in slightly acidified Ringer's solution, was injected into the external jugular vein. Very small doses (e.g., 1 c.c. of $\frac{1}{4}$ per cent.) produced an increase in the bladder volume without abolishing the undulatory contractions. When larger doses were given (fig. 4, Part III) these were abolished as well and the immediate effect then exactly resembled that of dividing the cord. This effect could be obtained with doses insufficient to abolish the corneal reflex. When the animals lived long enough there was a tendency for the volume to recover to some extent and for the undulatory contractions to reappear (fig. 4, Part IV). The

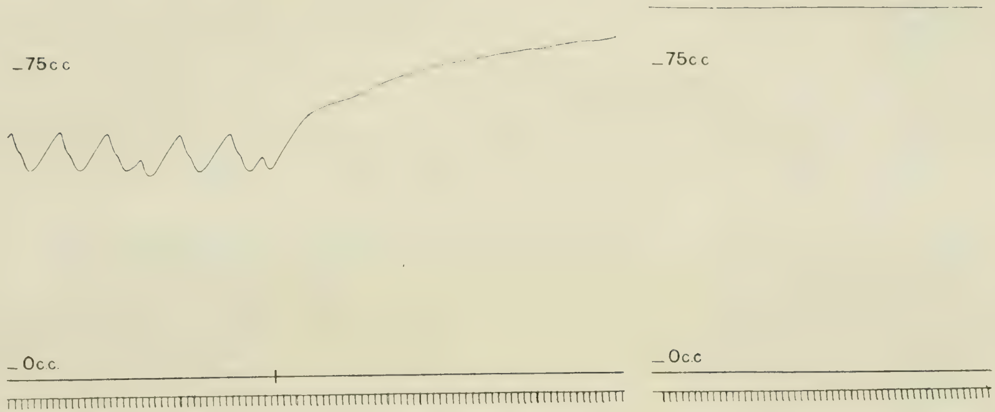

FIG. 4, Pts. IV and V.

Cat, male ; decerebrate. Bladder volume at 10 cm. H_2O pressure. Time marker, five seconds.

Part I : The fore-brain had been removed and the anæsthetic stopped two hours and twenty minutes before the beginning of the tracing. During the signal ether was given ; the corneal reflex was absent two minutes after the ether was started.

Part II : Commencing twenty-nine minutes after the end of Part I. During the signal chloroform was given, the corneal reflex was absent after two minutes, and after three, when the chloroform was stopped, respiration ceased ; artificial respiration was then performed for the next three minutes.

Part III : Fifty-five minutes after Part II. Artificial respiration : 2 c.c. of 2 per cent. curare were given subcutaneously fifteen to sixteen minutes before without obvious effect. During the signal 2 c.c. of 2 per cent. curare were given into the external jugular vein.

Part IV : Forty-eight minutes after Part III. At the signal the medulla was divided between the superficial origins of the hypoglossal and first cervical nerves.

Part V : One hour fifteen minutes after Part IV.

final volume was, however, considerably greater than the original one.

Curare was used in the series of seven experiments made to determine the level at which the motor tone of the bladder arises. The object of its use was to eliminate the disturbances in the bladder volume due to contractions of the abdominal muscles occurring in the powerful movements which always occur in a "spinal" cat. It is most important to know whether the volume alteration commences at the moment of division, in which case it is most likely directly due to the division, or a short time after, in which case it may be a secondary result produced by hæmorrhage: if curare is not used it is at this particular time that the muscular contractions are strongest and most frequent. The curare was given subcutaneously in doses of about $1\frac{1}{4}$ c.c. of 1 per cent. solution per kilo; more than an hour was allowed to elapse before proceeding with the experiment. Subcutaneously curare seemed less toxic and to produce its own characteristic effects just as well. The bladder volumes, in experiments where it had been given, seemed on the whole greater than in those where it had not, as would be expected from the result following its injection intravenously. In three of the seven experiments sections were made of the pons in its upper half; in all the volume recovered and undulations started again in two and were not abolished in the third; in two of these the section was not quite anatomically complete. The third was the lowest section; it went just in front of the superficial origins of the fifth nerves below and through the posterior parts of the inferior colliculi above. In all the seven experiments sections were made which produced a persistent dilatation which failed to pass away; the levels of these were 4 mm. below the upper border of the medulla, through the origins of the sixth nerves (two experiments), between the medulla and pons, through the lowest part of the pons (two experiments), and through the middle of the pons (fig. 5). Therefore, the highest section which gave the result and the lowest one which did not were both about the middle of the pons. From this it can be concluded that the tonic influence the central nervous system exercises on the bladder through the pelvic nerves arises in the central nervous system at about the level of the middle of the pons.

(2) Reflex Micturition.

A series of eighteen experiments was performed in the following way on female cats. The fore-brain was removed under chloroform as previously described and the chloroform administration stopped immedi-

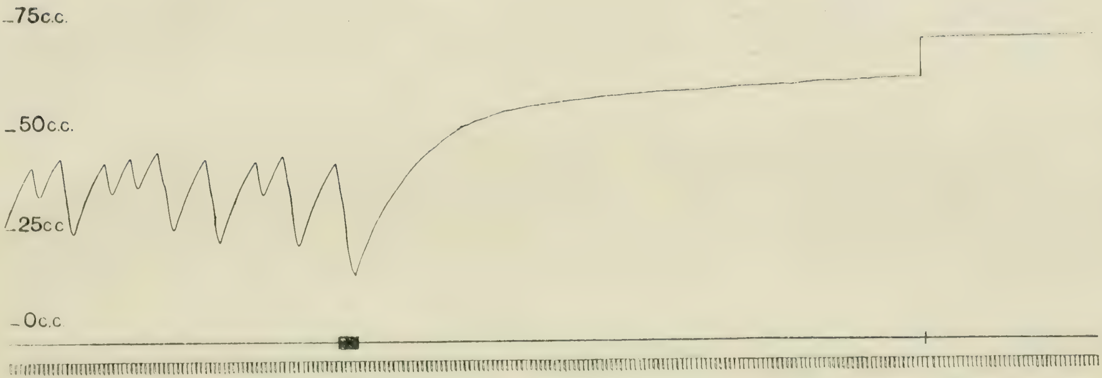

FIG. 5.—Cat, male. Bladder volume at 10 cm. H_2O pressure. The brain had been divided through the inferior colliculi, chloroform stopped, and the brain in front of the division removed two hours three minutes before the beginning of the tracing: 3 c.c. of 1 per cent. curare were given subcutaneously twenty-eight minutes after removal of the brain. At the first signal the brain was divided through the posterior part of the inferior colliculi above and the middle of the pons below. At the second signal the drum was stopped twenty minutes. Time marker, five seconds.

ately after. More than half an hour was then allowed to elapse before proceeding with the experiment. The cat was placed on its side at the end of a warm box inclined at about 15° to the horizontal, so that any fluid passed by the urethra ran on to a gutter at the end of a box into a receiver underneath, from which it could be measured. A brass catheter (size 7 Charrière) was then passed and the bladder emptied of any urine it might contain. In the earlier experiments a median incision was made dorsalwards into the perineal body to facilitate passing the catheter; this was afterwards abandoned as it was found unnecessary and also because it was suspected that the reflex might be interfered with by the irritation necessarily set up. The catheter was then connected, by means of a rubber tube, fitted with a Mohr's clip, to a warm water reservoir about 50 cm. above it; a metal worm in a warm water bath was interposed between the reservoir and the catheter to eliminate some of the cooling certain to occur in the tubes between the two. To make an observation the clip was removed from the rubber connecting tube and the vulval opening watched; when a definite escape of water at the side of the urethra had been established, e.g., after the escape of four to six drops, the tube was clamped off and the catheter removed from the urethra, the quantity of water passed was measured and the catheter passed again after to determine the quantity remaining in the bladder, if any. This was repeated several times in each experiment.

It was found that the water was passed in a continuous stream at first and often finished with a short series of jets; immediately after the passage of water had stopped one or more strong contractions of the perineal muscles usually occurred. The commencement was often initiated by an abdominal contraction of sufficient force to move the limbs and arch the back; this sometimes occurred just before and sometimes just after the withdrawal of the catheter. After the first observation had been made the volume necessary to provoke the reaction was more or less the same in each individual in subsequent observations. It was nearly always less than the volume of urine passed at a normal micturition and often very many times less, for instance, in seven out of the eighteen experiments it was constantly less than 20 c.c. The rapidity of filling the bladder, the fact that, though warm, the water was probably never exactly at the body temperature, and possibly also the presence of a catheter in the urethra, obviously may be sufficient to account for this deviation from the normal without the supposition that with the removal of the fore-brain there has been a removal of inhibition,

though as will be shown later there is a certain amount of reason to believe this also occurs. In all observations of eleven out of the eighteen experiments the amount of water left in the bladder at the end of the act was either none at all or less than 1 c.c. In three others it was $1\frac{1}{2}$ to 3 c.c., in the first observation in each, and less than 1 c.c. in the subsequent observations in each. In one other it was $1\frac{1}{2}$ to $2\frac{1}{2}$ c.c. in each observation. In these fifteen experiments therefore the micturition reflex was as nearly perfect as it is likely to be found. The remaining three all had very appreciable quantities remaining in the bladder at the end; an incision had been made into the perineum in all three cases; all three showed very strong abdominal contractions at the commencement of the act and the initial stream of water had an explosive force but stopped quite suddenly as if the urethral muscles had closed too soon. It is possible that the perineal incision may have had something to do with the unusual phenomena seen in these three experiments; it was made in eight of the other fifteen experiments, so this does not seem very probable. It seems much more likely that pressure on the remaining part of the brain from hæmorrhage into the posterior fossa of the skull was the cause, since the same effect was sometimes seen, in experiments where the passage of water had previously been normal, after interference with the posterior fossa or its contents. In one of the three experiments 5 to 6 c.c. were passed at each observation and the quantity left in the bladder gradually rose, being 11 c.c., 23 c.c., and 41 c.c.; during the fourth observation the cat developed spasmodic inspirations, scratch movements followed and then respiration ceased, while the heart continued beating; a thin clot was found on the ventral surface of the medulla and pons; it must be admitted however that this can sometimes be found in cats after decerebration in the way described which have not had any symptoms of pressure in the posterior fossa. In the second 4 c.c. to 8 c.c. of water were passed at each observation and the water remaining in the bladder was 17 c.c., 12 c.c. and 8 c.c. respectively: the cat died less than half an hour after the third observation and clots were present as in the last one. In the third experiment the residual water was relatively less and diminished with time, till in the last observation 62 c.c. were passed and 10 c.c. remained in the bladder; the cat died soon after this but the cause of death was uncertain as respiration stopped after some spasmodic inspirations five minutes after the injection of 5 c.c. of 10 per cent. cocaine hydrochloride into the bladder, an amount subsequently found sufficient to produce at any rate very severe symptoms. Whatever

the cause of these three exceptions the facts stated seemed quite sufficient to justify the conclusion that reflex micturition takes place normally in cats in the absence of the whole of the fore-brain and anterior part of the mid-brain.

Chloroform seems to have a very depressing effect on reflex micturition even when the anæsthesia is light. In eight of the above experiments an observation was made while the cat was under chloroform and before the fore-brain had been removed. In all the eight a very much larger quantity of water had entered the bladder before it escaped from the urethra than occurred after decerebration when the anæsthetic had been stopped some time. In all the eight a very considerable amount remained in the bladder after the escape from the urethra had stopped; in six this was more than twice the amount passed. Instead of being passed in a forcible stream the water came away in drops in these experiments, which were sometimes fast enough to form a continuous stream at the very beginning but never later. In another experiment, after decerebration, four observations were made and 8 c.c. passed in each one with a residual of nothing to five drops: chloroform was then given and the observation repeated, 7 c.c. were passed and 78 c.c. remained in the bladder.

In six of the experiments observations were made after injecting cocaine hydrochloride solution into the bladder. The solutions were left in fifteen minutes or more. As already stated, general symptoms are easily produced by this with strong solutions (e.g., 5 c.c. of 10 per cent.). The symptoms are a marked increase in respirations and an increase in the rigidity, particularly as shown by head retraction.

Five c.c. of 1 per cent. solution in one experiment had very little, if any, effect. In the remaining five intermediate strengths or quantities were used; they all required a greatly increased volume of water to provoke an escape of it from the urethra, and all had a very considerable volume left in the bladder after the passage of water had stopped. These results agree with those of Shattock [10] with cats under A.C.E. mixture. In all cases the effects were very transitory.

In a few cases the bladders were examined with X-ray plates after filling them with 10 per cent. colloid silver or a suspension of bismuth carbonate. In no case was a shadow present between the internal urinary meatus and the compressor urethræ unless the solution had actually been passed while the plate was being taken, and this was so whether the bladder had previously been injected with cocaine or not. Hence the solution is held at the internal meatus in decerebrate cats,

and not, as in those which have had a transection of the cord, at the compressor urethræ.

In two of the experiments the cord was divided at its union with the medulla and in one the medulla at the apex of the calamus. Observations made after showed the following changes. Firstly, the water did not escape from the urethra till the bladder contained a greater volume than that which was necessary to provoke it previously. Secondly, the amount passed after withdrawal of the catheter was smaller and instead of being passed in a forcible stream came away in a series of drops which sometimes showed rhythmic accelerations corresponding with visible rhythmic relaxations and contractions of the perineal muscles. Thirdly, when strong perineal contractions at the end of the act had been present before the division, they were absent after. Fourthly, a large amount of water remained in the bladder after the passage from the urethra had stopped. Reflex micturition was therefore abolished in these experiments, from which it may be concluded that it depends on the integrity of some part of the brain between the apex of the calamus and the mid-brain and on the connection of this part with the central nervous system below it. Reflex micturition had been shown by Elliott [4] to be abolished by destruction of the brain; he suggested that this effect was due to shock. Other reflexes, however, do not seem to be similarly affected; the shock can hardly be greater than that of removal of the fore-brain, and further, as stated in a previous paper [1], it could not be obtained some time (five weeks) after the cord had been divided. In three of the experiments the cerebellum was removed; post mortem it was found in all that the removal was incomplete, though the fragments remaining were small. In one of these experiments there were no changes in the reflex micturition after the removal, except that the bladder only tolerated about half the distension it did before. In the second the water was passed in a series of forcible jets instead of in a stream in the earlier observations after removal of the cerebellum, and in the later ones in drops only; in the earlier observations little or no water was left in the bladder at the end, while in the later ones there was a very considerable quantity. In the last the water was passed in a very forcible stream accompanied by a strong contraction of the abdominal muscles; the stream usually stopped suddenly and there was always a large amount of water left in the bladder; this cat developed spasmodic inspirations about an hour and twenty minutes after removal of the cerebellum. It seems probable, therefore, that removal of the cerebellum has no effect

on reflex micturition and that the effects of the operation, when any are present, are due to other parts of the brain below the tentorium being affected by hæmorrhage. In another experiment an incomplete division at the level of the upper part of the pons was made ; spasmodic inspirations developed thirty-six minutes later, and reflex micturition during this time showed the changes described in the last two experiments in which the cerebellum had been removed.

(3) The Reflexes composing Reflex Micturition.

The first fifteen experiments made to analyse reflex micturition into its component parts were performed as follows : Decerebrate female cats which had come round from the chloroform were used and a catheter passed as in the experiments on reflex micturition. The bladder was distended from a burette of warm water, so that known amounts in known times could be run in. Interposed between the burette and the catheter were : (1) a metal worm in a warm-water bath next the catheter ; and (2) a vertical tube, serving as a water manometer next the burette. Eighty cm. of the manometer tube contained 1 c.c., so pressure alterations produced no appreciable volume alteration. The bladder pressure could, of course, only be read when the clip of the burette was shut.

In these experiments it was found that active contraction of the bladder could be produced in one of two ways, by distension of the bladder to a given volume or by slightly moving the catheter in and out in the direction of the urethra.

To investigate the contraction resulting from distension, equal volumes of water, usually $2\frac{1}{2}$ to 10 c.c., were run into the bladder at definite intervals of time, usually every other minute, and the pressures noted at the end of one minute after each increase of volume. When a certain volume was arrived at it was found that the bladder pressure rose with or without an abdominal contraction, and this was usually accompanied by an escape of the water from the bladder at the side of the catheter ; the catheter was then disconnected to empty any bladder contents that might not have escaped, and the same series of operations repeated several times. The volumes tolerated by individual cats varied considerably, but after the first reaction had been produced the subsequent ones in the same cat occurred at more or less the same volumes, provided the distension had been made at the same rate. The increases of pressure produced by increases of volume within the limits of toleration were not great, but when that limit was reached the

pressure would at once, or in a few seconds, rise to three to fifteen times its previous value and gradually fall with the escape of the water from the urethra. It was noticed that the greatest height to which the pressure rose could be seen, at any rate often, to occur after the escape of the water had become well established; this is probably accounted for by the next reflex to be described. In some cases the maximum rise of pressure was preceded by one or more rises, each one larger than the one before; this may have been due to the catheter causing obstruction to the urethra, so that the first increase was incompetent to start micturition. There can be no doubt about the increase of pressure seen in these experiments being due to an active contraction for the following reasons. Firstly, though in the case of a non-living elastic bag a given increment of volume may give a larger pressure increase if it is applied when the bag is tensely distended than if it is applied when the bag is lax, it could not give an increase of 200 to 1,400 per cent. when the last equal increment of volume had given rise to hardly any pressure increase. Secondly, though in most cases the reaction naturally occurred when the water was actually being run in, in quite a large number it did not occur till after the clip of the burette was shut, and then the whole rise of 200 to 1,400 per cent. was actually seen. Thirdly, in the case of a non-living elastic bag, however great the pressure increase resulting from an increase of volume, it cannot exceed that at which the fluid is run in, whereas in the present experiments when the reactions occurred while the fluid was being run in, it often happened that the monometer pressure was greater than that in the burette, and water flowed back into the latter. This reflex contraction of the bladder resulting from distension occurred in four experiments where curare had been given which eliminated all abdominal contraction; the pressure rise was not quite as marked in these experiments, and there was always an appreciable volume of water remaining in the bladder after the reaction had finished, which is what would be expected from what has already been stated about the effect of curare on the motor tone of the bladder. In one experiment the effect was obtained after division of the brain through the upper part of the pons. In five experiments the medulla was divided at various levels between its upper border and the apex of the calamus; the effect was the same in all. The distension being carried on at the same rate as before making the brain section, it was found that the pressure given by a volume just below that which previously produced the reaction was approximately the same as that given by the same

volume before the brain section. When the distension was increased, it was found that the bladder tolerated a greater volume than before, and that the pressure rose gradually with each volume increment till escape of water from the urethra finally occurred; after a small amount had escaped the escape stopped, the pressure dropping slightly but remaining higher than it had been at any time before the brain section was made apart from the rise with the reaction. After the brain section no rise of pressure as high as the reactionary one could be obtained by slow increments of volume, because whenever the pressure rose above a certain amount escape started from the urethra, tending to bring it down again; at the same time a higher pressure could be maintained after the section than before, since the corresponding volume would have evoked the reaction before the section and caused emptying of the bladder. In eight of the fifteen experiments cocaine was injected into the bladder; the effect of this on the bladder was exactly the same as that of dividing the medulla; the bladder tolerated a greater volume than before, and no sign of a reactionary contraction was observed. After both cocaine and division of the medulla rhythmic alterations in pressure were sometimes seen over and above those due to respiration; these had no resemblance to the reactionary contraction with gradually increasing distension, because firstly they were much less in degree; secondly, they were more or less equal and quite regular for the same volume; and lastly, they usually diminished and finally disappeared as the volume was increased up to that giving a pressure which caused escape from the urethra; in no case were they increased by increasing the volume to this degree. In three experiments cocaine was injected into the bladder after division of the medulla; it then had no effect either on the final volume tolerated or on the pressures corresponding to the various volumes retained. These results differ from those of Shattock [10], who found the intravesical injection of cocaine was followed by an increased toleration to distension both in pithed cats and in those under A.C.E.

The second way in which active contraction of the bladder was evoked was by withdrawing the catheter a fraction of a centimetre and then pushing it back to its original position; the stimulus of a smooth foreign body moving in the urethra would appear to be the same as a stream of urine passing through it. It was found that in intolerant bladders of low capacity an increase of pressure followed this with any degree of distension, but in bladders of larger capacity a certain degree of distension was necessary for its production and

that the greater the distension the greater the rise of pressure evoked by this form of stimulus. The rise of pressure produced in this way was on the whole not quite as large as that produced by distension, the largest seen being thirteen and a half times the original; in many cases, it was, however, quite enough to start reflex micturition, the bladder contents being passed at the side of the catheter: as in the first reflex, this one can be obtained after curare and after division of both hypogastric nerves. In three of the experiments the pudic nerves were divided. In one the reflex did not appear affected by the operation. In the second it was certainly more difficult to elicit and when the rise of pressure did occur it was accompanied by a strong abdominal contraction. In the third the hypogastric nerves had previously been divided; a rise of pressure still occurred on moving the catheter after division of the pudic nerves, but it was so small as to raise doubts as to whether it was due to contraction of the bladder. After division of the medulla either no increase in pressure at all followed the movement of the catheter or it was small enough to be considered as due to the mechanical effect and not to bladder contraction at all. Injection of cocaine into the bladder abolished the reflex in some cases and not in others; anæsthesia of parts of the urethra by this means is probably fortuitous, so if the reflex arises from part of the urethra this is what would be expected. From these results it was concluded that in micturition there are two reflexes leading to a powerful contraction of the bladder; one arises from distension of the bladder and the other from urine running through the urethra; both reflexes are destroyed by transection of the medulla.

For the purpose for which it was used the method just described is open to three objections. Firstly, as the results themselves show, at any rate under certain conditions, the presence of a catheter in the urethra is a disturbing factor. Secondly, the movement in and out of the catheter itself may produce a pressure alteration which is unknown. Thirdly, though it is improbable, the second reflex contraction described might be due to the end of the catheter touching the wall of the bladder and not to the catheter stimulating the walls of the urethra. For these reasons and to examine the efferent side of the urethral reflexes another method was used.

An examination of the distribution of the branches of the hypogastric plexus to the bladder and urethra showed that it appeared anatomically possible to divide the bladder at its neck, i.e., just

proximal to where the urethra begins to expand, and tie a cannula into both cut ends so that the pressure of urethral resistance and of bladder distension could be observed simultaneously.

The method consisted in making a median suprapubic incision, and exposing the ventral surface of the neck of the bladder. A ligature on a curved needle was then put round it without dissecting its sides or dorsal surface and taking care to keep the needle close against the bladder, an incision was made into the neck distal to the ligature and a glass cannula tied in. A second thread was then passed in the same way distal to the incision in the bladder neck and a second cannula, which except in the earlier experiments was filled with water, tied into the cut end continuous with the urethra. The part of the circumference of the bladder neck not already divided was then cut through so that the two parts were completely separated. A metal worm in a warm-water bath was interposed between each cannula and its manometer. The water manometers used were vertical tubes with a T-piece at the lower ends fitted with clamps; they could be filled by connecting a burette to the ends of the T-pieces not connected with the cannulæ. The tube of the urethral manometer was wide (1 c.c. = 3 cm.), so that an escape of water from the external meatus should not cause a very rapid fall of pressure. The tube of the bladder manometer was narrow (1 c.c. = 34 cm.), so that a large pressure alteration should not cause a very marked volume alteration. Cats of both sexes were used, but females were found more satisfactory, partly, no doubt, because the anterior part of the urethra which is useless for micturition is absent, and probably partly because the urethral resistance at the compressor is less than in the male.

Five reflexes were found by this method, three leading to contraction of the bladder and two leading to relaxation of the urethra. The three leading to contraction of the bladder were those already described and will be considered first.

The reflex contraction of the bladder provoked by its own distension was examined in the same way as in the previous series of experiments and the results obtained were similar. The bladder was distended by running in equal volumes of water (2½ to 10 c.c.) every other minute and the pressure noted a minute after each increase of volume. The volumes at which reaction occurred were about the same for each individual, provided the rate of distension was the same, but varied considerably in different individuals. The volume just below that

which provoked the reaction gave an intravesical pressure which was always less than 10 cm. and sometimes less than 5 cm.; the height of the reactionary contraction gave a pressure from 40 to over 90 cm., the highest pressure which the manometer used would record. The contraction usually took place while the water was actually being run in, but not infrequently it took place a few seconds after the volume increase had finished and then its whole extent could be actually seen. It is not implied from this that the stimulus is a factor of the volume, it is clearly a factor of the intravesical pressure and not volume, since a reaction can be provoked by a much smaller volume if it is run into the bladder fast than by that which is necessary if run into it slowly: the same conclusion was arrived at by Mosso and Pellacani [9] in an entirely different way and as a clinical observation it is probably much older still. The few experiments which deviated from this description were clearly due to interference, probably by hæmorrhage, with the brain in the posterior fossa and will be described after the conditions which destroy the reflex have been considered. The reflex can be obtained after division of both pudic nerves or both hypogastric nerves: both afferent and efferent paths are therefore by exclusion in the pelvic nerves as was originally shown by Guyon [8]. The reflex was not obtained if the pelvic nerves were divided, if the spinal cord was divided either in the lower thoracic or the upper cervical region, or if the interior of the bladder was cocainized. The bladder on distension in all three cases behaved in the same way, the only reservation to be made being that in the case of cocaine the complete effect only lasts a few minutes; it is therefore necessary to run in such volumes every minute that the whole observation may be completed quickly after the cocaine has been let out of the bladder. The pressures corresponding to volumes tolerated before any of these three operations were performed were not less for the same volumes after performance of the operation. When the bladder was distended further, the pressure increased with each volume increment, the increases of pressure becoming greater for each volume increment the greater the distension of the bladder. No sign of a reactionary contraction was ever seen and the only thing that could possibly be mistaken for it was the presence in some experiments of the undulatory contractions already described; in addition to the reasons already given showing that these are not reflex contractions they were found to be present in some cases after division of both pelvic nerves. The statement that the pressures given by volumes tolerated before division of the cord or pelvic nerves are not less for the

same volumes after may at first sight appear at variance with the results already described and tracings shown in the section on the motor tone of the bladder. These experiments were, however, made at a pressure of 10 cm. and as already stated the bladder of a decerebrate cat reacts before this pressure is arrived at: the bladders in the volume experiments were therefore probably in a continual state of reaction and the sharp undulations shown in the curves really represent reactionary contractions. This conclusion is confirmed by the fact, already stated, that the most marked volume effects following these operations were found to occur when marked sharp undulatory contractions were present. From these results it is concluded that the reflex contraction of the bladder resulting from distension has both its paths in the pelvic nerves and that the paths down the central nervous system from the level of origin of the motor tone of the bladder to the origin of the sacral roots forming the pelvic nerves are essential to its existence. The experiments mentioned, where the behaviour of the bladder differed from that described, either showed the same volume and pressure relations as if the cord had been divided or with low volumes powerful irregular contractions were present as shown by considerable irregular increases of pressure; as the volume was increased these contractions usually got greater, but from the fact that the pressure they started from was so irregular it was impossible to say that any one of them was a reactionary contraction. Both these conditions appear to be due to failure of the functions of the lower part of the brain, the second being the early stage and the first the final one. The reasons for concluding this are, firstly, that in some experiments the bladder first reacted in the usual way, later the powerful irregular contractions set in, and finally it behaved as if the cord had been divided; some time after this respiration ceased while the heart continued beating, this having been preceded by spasmodic inspirations and these by rapid respiration with an occasional deep inspiration. Secondly, in those experiments where all stages were not seen but where the abnormality occurred, respiratory failure developed in the same way later. From the fact that the bladder motor tone centre is higher than the respiratory centre, the bladder would be expected to be affected before the respiration in decerebrate animals.

The second reflex leading to a powerful contraction of the bladder was evoked by running water through the urethra. The rise of pressure so produced was about the same degree as that in the last reflex described. It was investigated by distending the bladder to a volume

below that which had previously been found to produce a reflex contraction and after waiting a few minutes for the immediate effect of distension to pass off the pressure was read and noted at short intervals. The urethral stimulus was applied by gradually increasing the pressure in the urethral manometer till it became just sufficient to overcome its resistance, water would then escape from the external meatus and the pressure in the urethral manometer would fall in consequence. After an appreciable latent period of about two seconds the bladder pressure began to rise, in several experiments it rose to over 90 cm. The pressure remained up, though not at its highest, until the escape of water from the external meatus stopped. The contraction of the bladder occurred in these circumstances even when it was practically empty, since when the bladder cannula was disconnected from its manometer a few drops of water could at times be seen to be expelled from its open end after the escape of water from the external meatus had started. This reflex was not affected by division of both hypogastric nerves. It could be obtained after cocainizing the interior of the bladder, showing that the bladder motility is not impaired by cocaine and therefore that the abolition of the reflex contraction resulting from distension described in the last paragraph must be due to sensory loss. The reflex was abolished by division of both pelvic nerves, or division of the spinal cord either in the lower thoracic or upper cervical regions. From these facts it may be concluded that the efferent path of this reflex in the pelvic nerves and, as in the first reflex, that the paths from the middle of the pons for the motor tone of the bladder must be intact for its existence. In nine experiments the pudic nerves were divided after this reflex had been obtained, the pelvic and hypogastric nerves being intact: in eight the reflex could not be obtained after, but in the remaining one it could; in this experiment it was abolished by subsequent division of the hypogastric nerves. Afferent fibres for this reflex must therefore be constantly present in the pudic nerves: whether these are only exceptionally present in the hypogastric or whether they are constantly present and were usually destroyed by the operative precedures on the neck of the bladder was not decided.

In making the experiments just described a reflex rise of intravesical pressure was observed which was easily distinguished from that just described and which was first believed to be due to some extravesical cause. When the pressure in the urethral manometer was being raised, as the pressure equal to the urethral resistance was

approached sharp transitory rises of intravesical pressure amounting only to a few centimetres were very frequently observed. The stimulus of this reflex was evidently distension of the posterior urethra, since in one experiment as the urethral pressure was gradually raised it fell $\frac{1}{2}$ cm. each time it reached 30 cm., though there was no escape of water from the meatus till it was raised to 50 cm.: when it fell this $\frac{1}{2}$ cm. the bladder pressure immediately gave a small transitory rise and such rises continued after. This reflex rise of intravesical pressure was, however, seen to its greatest extent if instead of gradually increasing the urethral pressure this was suddenly increased by removing the clamp when the pressure in the manometer was just above that previously found necessary to force the sphincter. In these circumstances water did not usually escape from the external meatus for some seconds, but immediately the urethral pressure was increased the bladder pressure rose, often 10 cm., and rapidly fell again at once, the rise and fall taking about five seconds. That this rise of pressure was due to a bladder contraction was demonstrated by exposing the bladder through a lumbar incision and seeing it contract. This reflex is present after division of the spinal cord, both pelvic nerves, or both pudic nerves, but is abolished by division of both hypogastric nerves or the lumbar splanchnic nerves. Both paths of the reflex are therefore in the hypogastric nerves.

The fourth and fifth reflexes found by this method lead to relaxation of the urethra; they were evoked respectively by running water through the urethra and by distending the bladder. The fourth reflex will be considered first.

If the bladder was empty, or only contained a volume less than that which caused a reflex contraction, and the urethral manometer was gradually filled by running in equal quantities of water at equal intervals of time, when a certain pressure was reached water escaped from the urethra, usually after a few preliminary contractions of the perineal muscles. The escape was rapid and continuous and was either uniform or showed rhythmic accelerations, but, at any rate in females, never stopped and afterwards began again. As a result of the escape of the water the pressure fell and, when the escape had stopped, remained constant at a considerably lower value. If now the same operation was repeated the escape from the meatus did not start again till approximately the same pressure as that which produced it previously was arrived at, when the escape started and the pressure fell to approximately the same value as before. The figures from the first

experiment of this kind made (on a female cat) may be quoted for the sake of clearness. The pressure was gradually raised till escape of water from the meatus started when it was 34 cm., in the next half minute it fell to $10\frac{1}{2}$ cm., when the escape stopped and in consequence the pressure remained constant; the pressure was then increased again by 5 cm. each minute, escape of water from the meatus started again when the pressure was between 30 and 35 cm., and in the next half minute fell to 11 cm., when the escape stopped and the pressure remained constant. The fact that pressures above 11 cm. and below 30 cm. were resisted by the urethral muscles when the water was not escaping from the meatus, and not when it was, can only be due to a reflex relaxation of the urethra while the water is flowing. This reflex is still obtainable after division of both hypogastric nerves. After division of both pudic nerves water escapes from the external meatus under a much lower pressure than previously, unless the pressure is very high the escape is slow; when finally the escape has stopped any increase of pressure, even 1 cm., causes the escape to start again at once. The reflex is therefore abolished by division of both pudic nerves. It is a curious fact that sometimes the pressure to which the urethral manometer fell at the end of the reflex was lower than that of the urethral resistance after division of both pudic nerves. In this connection it is well to remember that the pelvic nerves are known to send inhibitory and the hypogastric motor fibres to the smooth muscle of the urethra [11], so it is possible that they take part in the efferent side of this reflex; if this is the case no effect would be produced on the reflex by their division unless they contained its afferent fibres as well, since by the method employed the whole urethral resistance was examined and it is well known that by far the greater part of this is due to striped muscle supplied by the pudic nerves [3]. It has been shown previously [1] that the pelvic nerves exercise an inhibitory tone on the urethra and that the hypogastric nerves do not exercise a motor one, so if either of these nerves take any part in the reflex the pelvics would appear the more likely. Neither division of both pelvic nerves nor transection of the spinal cord, either in the lower thoracic or upper cervical regions, abolished this reflex, but they did modify it apparently in exactly the same way. After each of these operations the pressure the urethra resisted was found to be rather increased. As the pressure was raised contractions of the perineal muscles started if they were not present before, or became more numerous and stronger if they were. When the resistance was overcome the water escaped in a series of jets

with definite pauses between them which got longer as the pressure in the manometer fell, the result of which was that the fall of pressure took a longer time to be completed. The fall of pressure after the escape had started was usually less in degree than when the pelvic nerves and spinal cord were intact, at the same time it was quite easy to show that intermediate pressures were supported after these operations as before, therefore the reflex must still have been present. From these facts it must be concluded that both paths of this reflex are in the pudic nerves, with the possible reservation for the inhibitory fibres of the pelvics, already mentioned, taking a small part in it. The modification of the reflex in the same way by division of either the pelvic nerves or the spinal cord is possibly due to the fact that both these lesions abolish the reflex bladder contraction provoked by running water through the urethra. In describing the next reflex increased intravesical pressure will be shown to be associated with a reflex relaxation of the urethra. It is possible that the same may occur from contraction of the bladder even though it is empty. If this is the case the reflex contraction of the bladder which follows the running of water through the urethra would reinforce the direct reflex relaxation just described and division of either both pelvic nerves or the spinal cord would abolish this reinforcement. In a few experiments the effect on this reflex of injecting cocaine into the bladder was found to be of the same kind as that of dividing the pelvic nerves, but it was never of the same degree.

The fifth reflex was demonstrated by filling the urethral manometer to a pressure intermediate to that which was previously found to start an escape of water from the external meatus and that to which it fell when this escape occurred. The bladder was then filled by running in equal amounts every other minute as before. Directly the reactionary rise of intravesical pressure took place water started to run from the external meatus, and the pressure in the urethral manometer rapidly fell in consequence. The pressure to which the urethral manometer fell when a reflex was provoked by this means was usually lower than that to which it fell when water was simply run through the urethra with the bladder empty as described for the demonstration of the fourth reflex; this was presumably because the fourth reflex was actually occurring as well as the fifth and so reinforced the effect of the latter. The same effect was produced if both hypogastric nerves had been divided. Division of both pelvic nerves abolished the reflex. It was also abolished if 5 c.c. of 4 per cent. cocaine hydrochloride was injected into the bladder and left there twenty minutes or more; this effect was

not very easy to observe on account of the transitory nature of the effect of cocaine already mentioned. Division of the spinal cord in either the lower thoracic or upper cervical region did not abolish the reflex. After this operation the volume which previously caused a reflex contraction of the bladder had no effect on the urethra, and it was not till a greater increase in the distension of the bladder leading to a considerable elevation in the intravesical pressure had been produced that water escaped from the meatus and the pressure in the urethral manometer fell. When this had started, each subsequent increase of volume, leading of course to a still greater intravesical pressure, usually caused a further escape of water from the meatus and further fall of pressure in the urethral manometer. If the urethral pressure which just caused the escape of water from the meatus was determined by the method of gradual increments from zero already described when the bladder pressure was considerably raised by distension and then again after the bladder had been emptied, it was found that a greater pressure was necessary to force the urethra with the empty bladder than with the distended. From these facts seen after transecting the cord it is evident that the stimulus depends on the tension of the bladder wall and not on the degree to which it is stretched, and further that the reflex is continuously acting so long as this tension remains sufficient to excite it. Distension of the bladder produced contractions of the perineal muscles like raising the urethral pressure, though not to the same extent as the latter; the escape of water from the meatus occurred in jets after division of the cord and these could be seen to be synchronous with the intervals between these contractions. The afferent path of this reflex must therefore be in the pelvic nerves. The efferent path must be in the pudic nerves, since after division of these no effect could be produced on the residual urethral resistance by distension of the bladder.

From the foregoing remarks it is evident that the five reflexes demonstrated by the method under consideration are of such a nature that when one starts the others are brought into action automatically. If, as seems probable, though no evidence has been brought forward to prove it, the first reflex to occur is the contraction of the bladder provoked by distension, this contraction will of itself lead to relaxation of the urethra through the fifth reflex described; the urine running through the urethra will by itself lead to a relaxation through the fourth reflex described, and after a latent period of two or three seconds it will also lead to a contraction of the bladder as long as the stream lasts through the second reflex. The importance of the second

reflex for emptying the bladder is evident, as if it did not exist the lowering of intravesical pressure due to the escape of urine would abolish the stimulus provoking the first reflex and so the contraction of the bladder would not persist to the end. The use of the third reflex, which depends on the hypogastric nerves, does not appear very evident.

After transection of the spinal cord the first and second reflexes are abolished but not the third, fourth and fifth, hence the escape of urine from the urethra of such animals is due to reflex relaxation of the urethra through the fourth and fifth reflexes, the feeble and transitory contraction evoked through the third reflex being incompetent for the purpose. The constant presence of residual urine in these circumstances is no doubt due to the absence of the first and second reflexes. The conclusion of Goltz [6] that micturition can take place after spinal transection was based on the assumption that the reflex escape of urine found by Giannuzzi [5] and himself to occur some days after this operation was due to reflex contraction of the bladder, but he described no observations proving that this in fact occurred. To make certain that the stage of "spontaneous micturition," now so well known to occur in animals some days after a spinal transection, was not due to the reappearance of a reflex contraction of the bladder, the spinal cords of two female cats were divided respectively at the 8th and 9th thoracic roots under chloroform. After twenty-six and thirty-five days respectively, the cats were decerebrated and examined by the experimental method under consideration. In both of them the fourth and fifth reflexes were present. No sign of a reflex contraction of the bladder could be obtained by distending it. On raising the pressure in the urethral manometer, a moderate volume being present in the bladder, sharp transitory increases of bladder pressure up to 6 cm. took place both before and at the time water began to escape from the external meatus, but no sustained or greater rise of pressure was present. Hence, even when spontaneous micturition has become established, the two reflexes which lead to a strong and sustained bladder contraction are still absent.

The existence of these five reflexes affords a ready explanation for the fact that urine in normal cats, as shown by Griffiths [7], is held at the internal meatus, whereas in cats that have had a spinal transection, both pelvic nerves divided or the posterior sacral roots divided, it is held at the compressor urethræ [1]. The resistance of the urethra after division of the pudic nerves is greater than the pressure the bladder will tolerate without reacting: the greater part of this resistance, at any rate in females, must be due to the smooth

muscle of the posterior urethra, hence the posterior urethra will normally always be closed except when a reaction is occurring, i.e., during micturition. After any of these three lesions mentioned, however, the reaction of the bladder to distension is abolished, hence it allows its pressure to rise to a much greater extent without urine escaping; the posterior urethra can then be forced but the compressor being stronger will hold the urine for a longer time till the pressure has increased still further. The same effect should be produced by cocainizing the bladder, but as already stated this was not found to be the case; the explanation of this discrepancy would appear to be that during the time necessary to fill the bladder and take the X-ray the effect of the cocaine had, at any rate partly, worked off.

(4) Micturition in Decerebrate Cats.

The passage of urine in a forcible stream very commonly occurs five to ten minutes after decerebration; in such cases there is nearly always a distinct, though often small, quantity of urine left in the bladder. This residual urine, from what has been said of the effect of chloroform on reflex micturition, can be accounted for by the cats not having recovered from the chloroform at this time. This passage of urine is such a frequent occurrence after decerebration when there is any quantity of urine in the bladder at the time that it is impossible to doubt that it is due to the operation; from its frequency it appears to be due to the removal of an inhibitory influence arising in the part of the brain removed, and not to any secondary result of the operation such as bleeding into the posterior fossa.

Eight cats were decerebrated—all but one under aseptic conditions—and after arrest of bleeding the skull cavity was filled with paraffin wax by Bazett's method [2] and the wound closed. As it was necessary to tell when the cats passed urine, they could not be kept in a bath as was done by Bazett. They were, therefore, put in a closed cage in which a number of electric lights were fixed. The rectal temperature was taken periodically and the temperature of the cage altered when necessary by altering the number of lights burning. The experiments were all made in July or August. The duration of decerebrate life varied from 4 to 40 hours and averaged 16 to 17. The cage was placed on a tray which sloped towards its centre where it was perforated, a receiver placed under the perforations showed whether urine had been passed or not. In six of the eight cats micturition appeared to be performed normally. It

took place nineteen times and was actually seen in four of the nineteen. The urine was passed in a stream, finishing with a few jets associated with perineal contractions. Palpation of the abdomen after showed the bladder to be either empty or only to contain a small volume of urine such as might have accumulated in the interval of time since it was last looked at. The volume passed at each micturition varied from 10 to 120 c.c. and averaged 47 c.c., which is rather below the average quantity passed at a single micturition by a normal cat. The cat, which lived forty hours after decerebration, weighed 3,170 grm. and micturated seven times, the volume passed at each micturition varied from 34 to 70 c.c. and averaged 53 c.c. There, therefore, seems to be a slight degree of frequency of micturition in decerebrate cats, but the act of micturition itself is performed normally. In the other two cats micturition was not normal in that there always appeared to be residual urine about 5 to 10 c.c. in one and about 10 to 20 c.c. in the other. In both the amount of urine passed at each micturition was very small, 5 to 15 c.c. in one and 2 to 12 c.c. in the other. When micturition occurred it was passed in a stream in one, but it was never actually seen in the other.

REFERENCES.

[1] Barrington. "The Nervous Mechanism of Micturition," *Quart. Journ. Exper. Physiol.*, 1914, **8**, 33.
[2] Bazett. "The Use of Decerebrate Preparations for Experiments extending over Several Days," *Proc. of the Physiol. Soc.*, July 12, 1919, xlii.
[3] Courtade and Guyon. "Sur la résistance du sphincter vesico-uréthral," *Compt. rend. de la Soc. de Biol.*, 1895, 620.
[4] Elliott. "The Innervation of the Bladder and Urethra," *Journ. of Physiol.*, 1907, **35**, 367.
[5] Giannuzzi. (Quoted by Goltz.) *Rivista scientifica* (Siena), 1869, Anno I, Fasc. I.
[6] Goltz. "Ueber die Functionen des Lendenmarks des Hundes," *Pflüger's Archiv*, 1874, **8**, 460.
[7] Griffiths. "Observations on the Urinary Bladder and Urethra: III," *Journ. of Anat. and Physiol.*, 1895, **29**, 262.
[8] Guyon. "Rôle du nerf érecteur sacré dans la miction normale," *Compt. rend. de la Soc. de Biol.*, 1900, 712.
[9] Mosso and Pillacani. "Sur les fonctions de la vessie," *Arch. ital. de Biol.*, 1882, **1**, 313.
[10] Shattock. *Proc. of Roy. Soc. Med.* (Section of Pathology), 1909, **2**, 99.
[11] v. Zeissl. "Ueber die Innervation der Blase," *Pflüger's Archiv*, 1893, **53**, 560.

LESIONS OF THE POSTERIOR TIBIAL NERVE.

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AMONG the injuries to peripheral nerves resulting from gun-shot wounds, those most frequently overlooked, in the writer's experience, are lesions of the posterior tibial nerve. By the posterior tibial nerve is meant that part of the continuation of the internal popliteal nerve which lies between the lower border of the popliteus muscle above and the hollow behind the internal malleolus below. With injuries to the internal popliteal nerve itself (i.e., that portion of the internal division of the sciatic nerve lying above the lower border of the popliteus) this communication does not propose to deal; the resulting paresis of the calf muscles alone usually draws attention at once to the nature of the lesion. With the posterior tibial nerve, however, the case is different; the motor disability is relatively slight, more particularly when the lesion is below the origin of the nerve branches supplying the flexor longus hallucis and flexor longus digitorum muscles. The patient usually appears complaining of pain or numbness in the foot; it is seen that he is able to plantar-flex the foot at the ankle and also to flex the toes. The condition is often termed "functional" and the patient labelled "neurasthenia." Many of the sixty-four cases on which the following observations are based were received with such a diagnosis. When any penetrating wound of the calf or ankle has occurred, therefore, it is important to make a careful examination of the foot with regard to the presence or absence of sensory changes over the plantar area, and of paresis, atrophy or abnormalities of electrical reaction in the intrinsic muscles of the foot.

Lesions of the posterior tibial nerve, among nerve injuries generally, are somewhat uncommon. In 1,688 injuries to peripheral nerves, I met with sixty-four posterior tibial nerve lesions. This incidence is higher than that in both Purves Stewart's and Evans's series of 520 cases of nerve injury—four posterior tibial lesions (0·76 per cent.)—

and in Tinel's series of 639—eighteen posterior tibial lesions (2·8 per cent.).

To review briefly the anatomy of the posterior tibial nerve. From its origin at the lower border of the popliteus muscle, where the internal popliteal nerve ends, the posterior tibial nerve lies on the tibialis posticus muscle and the tibia, occupying with the posterior tibial vessels a sheath in the intermuscular septum which divides the superficial from the deep muscles of the back of the leg. During its course, the nerve supplies muscular branches to the flexor longus hallucis and flexor longus digitorum; the branches of supply to the two heads of gastrocnemius, soleus and tibialis posticus and the communicans tibialis are usually given off from the internal popliteal nerve in the lower part of the popliteal space. The soleus and tibialis posticus are often supplied by a common trunk, but occasionally the nerve to tibialis posticus may arise separately from the upper part of the posterior tibial nerve. As regards cutaneous branches, the internal calcaneal nerve is given off near the termination of the posterior tibial trunk; it pierces the internal annular ligament and is distributed to the skin of the heel and the posterior part of the sole. The posterior tibial nerve then terminates by dividing into internal and external plantar nerves beneath the internal annular ligament, the former nerve supplying flexor brevis digitorum, abductor and flexor brevis hallucis, the first lumbrical and the skin of the inner part of the sole as far as the mid-line of the fourth toe. The external plantar nerve supplies accessorius, the interossei, abductor and flexor brevis minimi digiti, the three outer lumbricals, adductor hallucis and the outer part of the skin of the sole from the mid-line of the fourth toe. The plantar collaterals supply, by means of dorsal branches, the dorsal surface of the ungual phalanges.

The internal plantar nerve is homologous with the median nerve of the hand and the external plantar with the ulnar.

I.—Syndrome of Complete Interruption.

The nerve fibres of the lower or peripheral segment of the nerve are entirely cut off from those of the upper or central end. This separation occurs for the reason that the nerve is completely severed, extremely compressed, or owing to the formation of a fibrous cicatrix as the result of tearing or bruising of the nerve.

Even when the nerve is wholly divided and the skin of the plantar area completely anæsthetic and analgesic, considerable pain in the sole

of the foot on walking may be the main symptom. The source of the pain is the end-bulb of the upper segment, the sensation being referred to its area of distribution, a point sometimes lost sight of. On the other hand, the complaint may be "numbness and weakness" in the foot, especially on exertion.

The physical signs of complete interruption are as follows:—

Sensory disturbances.—There is loss of sensibility to light touch (cotton wool) and pin-prick over the entire sole of the foot, the area of loss extending laterally to about half an inch beyond the margin of the

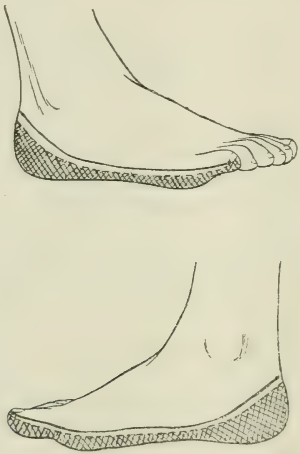

Fig. 1.—The area of loss of cutaneous sensibility with complete interruption of the posterior tibial nerve. The upper limits of loss of sensibility to cotton wool are represented by the continuous line; over the shaded areas sensibility to pin-prick is also lost. Loss of both cotton wool and pin-prick sensibility is continued over the entire sole of the foot. From a case (Case 1) in which at subsequent operation the nerve was found to be completely severed.

sole; the area of anæsthesia to light touch is, of course, slightly more extensive than that of analgesia to pin-prick. On the inner side of the foot the margin of both anæsthesia and analgesia is well defined; on the outer side adjoining the external saphenous area it is less definite. In some diagrams of the cutaneous area supplied by the posterior tibial nerve through its calcaneal, internal and external plantar branches, the lateral limits of the area are shown parallel to the margins of the foot throughout its length. I have invariably found, however, that in complete interruption of this nerve the cutaneous loss of sensation

involves a considerable portion of the lateral and posterior aspects of the heel (*vide* fig. 1). Owing to the relative thickness of the skin of the plantar area, sensation to cotton wool is often difficult to estimate, but this is not usually the case at the margins of the foot, to which particular attention should be directed. The dorsal surfaces of the outer four toes are usually described in posterior tibial nerve lesions as being insensitive to light touch, while no loss of sensibility to pin-prick is found over either their dorsal or plantar surfaces (fig. 2 A). While this distribution occasionally holds good, I have nevertheless found considerable variations. For instance, in one case in which subsequent operation showed the posterior nerve to be completely severed, loss of

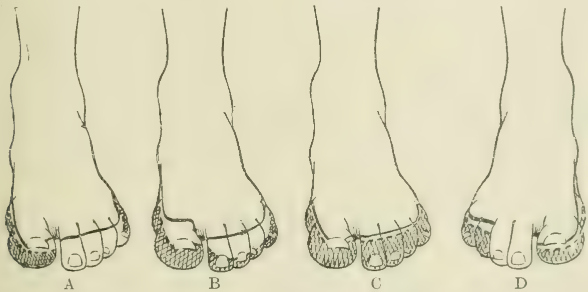

FIG. 2.—Illustrating variations in the loss of sensibility (to light touch and pin-prick) over the dorsal surface of the toes in complete interruption of the posterior tibial nerve. The continuous lines represent the upper limit of loss of sensibility to light touch (cotton wool); sensibility to pin-prick is also lost over the shaded portions. The loss to both forms of sensibility is continued round the anterior extremities of the toes to the plantar aspect in B and C and around the first, fourth and fifth in D. In A there is no loss of sensibility to pin-prick over the plantar surfaces of the outer four toes, but in D this form of sensation is lost on the plantar aspects of the third and fourth toes, but not of the second and third.

sensibility to pin-prick was present over the plantar surfaces of the toes and extended round their extremities to half way down the nails; loss of sensation to cotton wool was also present on the dorsum of the hallux, which is unusual (fig. 2 B). In a second case, the loss to pin-prick extended down the dorsal surfaces of the outer four toes as far as the proximal inter-phalangeal joints (fig. 2 C), and in a third case of complete division of the nerve, the outer two toes were insensitive to cotton wool and pin-prick stimuli both on their dorsal and plantar aspects, while the second and third toes exhibited normal sensibility on their dorsal surfaces, sensibility to cotton wool only being lost on the plantar (fig. 2 D). Also, in my experience, it is more usual to find

sensibility to pin-prick absent than present over the plantar aspect of the toes. In most cases I have found no loss of deep pressure sense; in two cases only was there apparent loss of deep sensibility over a small area of the plantar aspect of the heel.

Motor disturbances.—On inspection, it is found that there is usually hyper-extension of the proximal phalanges of the outer four toes and acute flexion of the second and third phalanges; this occurs owing to paralysis of the interossei which normally flex the toes at the metatarso-phalangeal joints and extend at the interphalangeal joints. The plantar muscles are paretic and wasted, the concavity of the plantar arch thus being exaggerated; in my experience, however, any marked degree of pes cavus is seldom met with. When the lesion lies below the origin of flexor longus hallucis and flexor longus digitorum, flexion of the toes is well performed owing to the action of these undamaged long flexors, but in many cases the degree of flexion is not so great as is possible in the toes of the normal limb; in others there is no difference. The degree of extension of the toes is often somewhat limited as a result of paralysis of the interossei and, for the same reason, they cannot be separated. The ankle-jerk is usually much diminished or is absent.

When the lesion lies above or involves the branches supplying flexor longus hallucis and flexor longus digitorum, in addition to the changes described above as resulting from complete interruption of the nerve at the lower level, voluntary flexion of the toes is impossible. The patient also has some difficulty in rising on the toes of the affected leg, but he is not entirely unable to do so.

Electrical changes.—In most works on medical electricity and nerve injuries, the motor points of the muscles supplied by the plantar branches of the posterior tibial nerve are not mentioned. They are therefore shown on fig. 3. Prior to testing the electrical reactions, the foot should be soaked in warm water for ten to fifteen minutes. In a complete lesion, on stimulating the posterior tibial nerve behind the internal malleolus with faradism, no response will be evoked in the plantar muscles. Similarly, when the active electrode is placed on the motor points of the various muscles supplied by the nerve, no contraction will occur. With galvanism, a response can usually be elicited in flexor brevis digitorum, abductor hallucis and in the interossei, the degree and nature of the contraction varying with the length of time that the nerve lesion has existed; in complete lesions of some months standing, the reaction is very sluggish and A.C.C. is greater than K.C.C.

With the condenser set—which I have invariably used for testing reactions in nerve injuries[1]—a response cannot usually be obtained at 100 volts in a normal flexor brevis digitorum and abductor hallucis with any stop under No. 6 (0·10 microfarads) and in normal interossei under No. 5 (0·083 microfarads) Lewis Jones scale. In complete lesions of the posterior tibial nerve, no response can usually be elicited in the affected muscles even with the highest stop—No. 12 (4·0 microfarads) at 100 volts.

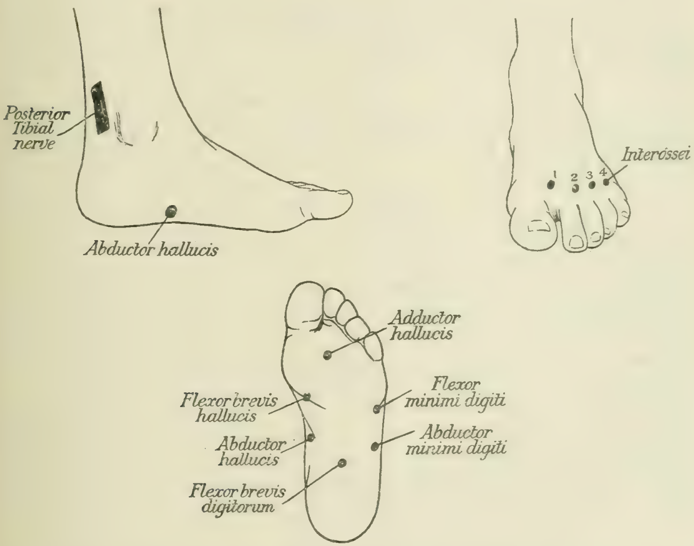

FIG. 3.—The motor points of the muscles supplied by the posterior tibial nerve.

In cases in which the flexor longus hallucis and flexor longus digitorum are involved, the response to faradism is absent in these muscles, and with the condenser a reaction will not, as a rule, be obtained with any stop below No. 10 (2·0 microfarads at 100 volts).

Trophic disturbances may be present in the form of scaly desquamation or mechanical ulcers over the sole of the foot; the latter lesions usually occur on the heel or at the base of the great or little toes.

[1] *Brit. Med. Journ.*, September 11, 1920.

Of the sixty-four cases of posterior tibial nerve lesions in my series, in sixteen interruption of the nerve was complete. Of these sixteen cases, in twelve the site of the injury was below the point of origin of the branches to flexor longus hallucis and flexor longus digitorum; in three it was above this level, and in the remaining case the injury involved the nerve at the level of the origin of the branch to flexor longus digitorum, the flexor longus hallucis, which is supplied from a slightly higher level, escaping and showing good voluntary movement and normal electrical reactions.

The following cases illustrate complete interruption of the posterior tibial nerve below the branches to the flexor longus hallucis and flexor longus digitorum muscles :—

Case 1.—C. McC., seen May, 1918, had received a bullet wound through the lower part of the leg in June, 1917; owing to sepsis, three months elapsed before the wound healed. No nerve injury was suspected. He was sent up for examination with the statement: "This man professes that he cannot carry on and no reason can be found." The complaint was one of numbness in the foot, a sensation as of pins and needles above the ankle and consequent difficulty in marching.

The scar of the entry wound was seen just behind the postero-internal border of the tibia, four inches above the internal malleolus, the exit scar being situated immediately external to the tendo Achillis, three inches above the external malleolus. There was cutaneous anæsthesia and analgesia over the entire plantar area. All voluntary movements at the ankle-joint were normal and the patient was able to flex the toes, the degree of flexion, however, being less than in the normal foot. Flexor longus digitorum and flexor longus hallucis responded normally to faradism, but no reaction was obtained in flexor brevis digitorum, abductor hallucis, nor in the interossei; these muscles also failed to respond even on No. 12 stop (4·0 microfarads) at 100 volts with the condenser set.

At operation, performed two days later, by Major Cecil Rowntree, the nerve was found densely adherent in firm fibrous tissue and, on dissection, was shown to be completely divided, an interval of half an inch separating the two extremities.

Case 2.—A. B., seen September, 1918, had been wounded by a piece of shrapnel in September, 1917, the missile entering three inches above the internal malleolus and leaving over the fibula, two inches above the external malleolus. The latter bone was fractured and owing to sepsis the wound had not healed until June, 1918, when he was discharged from the Army. No nerve injury had been diagnosed.

On examination, the patient complained of "burning pain," aggravated by walking, in the sole of the foot and above the back of the heel. There was

cutaneous anæsthesia and analgesia over the entire plantar area (fig. 2 B) and percussion over the entry wound gave rise to pain of a tingling character referred to the sole of the foot. All movements at the ankle-joint were normal; the toes could be well flexed but extension was somewhat limited. The plantar muscles were paralysed and atrophied. No response could be obtained with either faradism, galvanism or on the highest stop (No. 12—4·0 microfarads) of the condenser at 100 volts.

At the subsequent operation, the posterior tibial nerve was found involved in dense scar tissue, which, on being dissected away, revealed an upper bulbous extremity united to the lower segment of the divided nerve by a thin fibrous cord an inch long. After removing the upper end-bulb and incising the lower fragment until nerve fibres were seen, it was found impossible to bring the two severed ends of the nerve together. Consequently, the operator turned down a flap from the upper segment of the nerve, joining it to the lower—a useless procedure if regeneration is to occur. When the patient was examined eighteen months later, all the signs of a complete posterior tibial nerve interruption were still present, there being no evidence whatever of regeneration.

II.—Syndromes of Incomplete Interruption.

Incomplete interruption of the nerve functions may occur as the result of partial division, infiltration by interstitial traumatic hæmorrhage, or by involvement in and compression by cicatrical fibrous tissue or a fibrous band.

The sensory changes consist of loss of sensibility to cotton wool and pin-prick over the whole or only a portion of the plantar area, or, more frequently, mixed anæsthesia and over-reaction. The external plantar area is invariably the more anæsthetic, the internal plantar area often being the site of over-reaction. Varieties of the sensory changes that occur are illustrated by fig. 4.

In lesions of the inferior part of the nerve, there is more or less paresis and wasting of the plantar muscles and interossei, with diminished or absent reaction to faradism. With the condenser set, reactions are obtained on stops from No. 9 (1·0 microfarad) to No. 12 (4·0 microfarads) at 100 volts.

When the nerve has been damaged at the higher level, there is, in addition to the changes mentioned above, paralysis or weakness of the long flexors of the toes; in some cases voluntary movement in flexor longus hallucis is retained while flexor longus digitorum is paretic, and vice versa. In one of my cases, the lesion was complicated by a hysterical paralysis of the entire limb; the condition was easily differentiated by the extent of the paralysis and the faradic reactions.

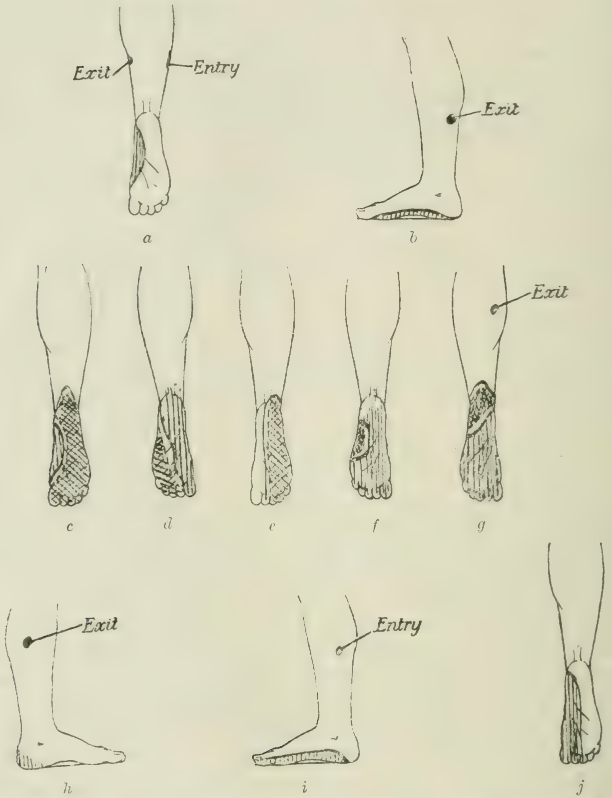

FIG. 4.—Varieties of the sensory changes encountered in incomplete interruption of the posterior tibial nerve. Over-reaction is represented by vertical lines. Shaded areas represent loss of sensibility to cotton wool and pin-prick, while the continuous lines just beyond shaded areas indicate the limit of loss of sensibility to cotton wool. a and b = over-reaction to pin-prick, with diminished sensation to cotton wool, over a portion of the internal plantar area (Case 5). c = Loss of sensibility to cotton wool and pin-prick over the greater part of the plantar area, a small area on the inner side (internal plantar) showing over-reaction. d = loss of sensibility to cotton wool and pin-prick over the external plantar area with over-reaction to pin-prick over the internal plantar and calcanean areas. e = Loss of sensibility to cotton wool and pin-prick over the external plantar and external portion of the calcanean area, the remainder of the area of posterior tibial nerve supply being normal. (At operation it was found that the outer side of the nerve was thickened and adherent to the adjacent tissues). f = A small area of loss of sensibility to light touch and pin-prick in the external plantar area, the remainder of the sole showing over-reaction. g = Loss of sensibility to cotton wool and pin-prick over the calcanean area, the external and internal plantar areas showing over-reaction. h = Over-reaction to pin-prick on the internal aspect of the calcanean areas. i and j = Over-reaction to pin-prick, with diminished sensation to cotton wool, over the internal plantar area, other areas being normal (Case 6).

The electrical reactions vary with the degree of the lesion; with faradism it will sometimes be possible to obtain a sluggish response in the long flexors but not in the plantar muscles or interossei. With the condenser, reactions are elicited on stops varying from No. 7 to 10 (0·25 to 2·0 microfarads) at 100 volts in the long flexors, and No. 9 to 12 (1·0 to 4·0 microfarads) in the intrinsic muscles of the foot at 100 volts.

In practically all cases of incomplete lesions of the posterior tibial nerve, there are symptoms of more or less irritation of the nerve trunk. The irritation gives rise to painful sensory disturbances varying from simple neuralgia to intense causalgia. In my experience, however, the posterior tibial nerve is less often the seat of true causalgia than the internal popliteal nerve. Spontaneous pains, described as shooting, burning or pricking in character, may be complained of. The pain is intensified by movement, muscular contraction, extremes of heat and cold, and by mechanical stimulation of the plantar area. In some cases there is cutaneous anæsthesia over the sole of the foot, but deep pressure gives rise to considerable pain; in others, diminished sensibility to light touch (cotton wool) coexists with over-reaction to pin-prick. Friction, firm tactile stimuli or pin-pricks applied to a plantar area of over-reaction results in a diffuse and radiating painful sensation of a burning or rending character, the stimulus being only imperfectly differentiated and localized by the patient. When, in an incomplete lesion of the posterior tibial nerve, there exists both over-reaction and anæsthesia over the skin of the plantar area, it is almost invariably the internal plantar that exhibits the over-reaction. This is illustrated by the following case, which is also of interest in showing over-reaction of the lateral part of the heel, which has previously been referred to as being within the area of posterior tibial supply.

Case 3.—A. M. C., seen April, 1918, was wounded through the ankle by a bullet in April, 1917, exactly a year previously. Since being wounded he had complained of pain in the instep and across the base of the toes, becoming more severe on attempting to walk.

On examination, the entry scar was seen an inch above and behind the tip of the internal malleolus, the exit being just above the external malleolus. The greater part of the plantar area was insensitive to both cotton wool and pin-prick, but on the inner side of the sole and heel there was an area showing marked over-reaction to pin prick but not to cotton wool (fig. 5). Flexion of the toes was well performed. The plantar muscles and interossei all failed to respond to faradism, and only reacted on No. 12 stop (4·0 microfarads) at 100 volts with the condenser set. A diagnosis of adhesion in fibrous tissue,

with incomplete interruption and slight irritation, was made and exploration advised.

At the operation performed a few days later by Major R. Jocelyn Swan, the posterior tibial nerve was found tightly compressed and bound down by a band of fibrous tissue in the track of the missile ; it was also adherent to the surrounding tissues and exhibited slight fusiform enlargement. Prior to freeing the nerve, faradic stimulation of its trunk above the lesion gave rise to a moderate response in the plantar muscles.

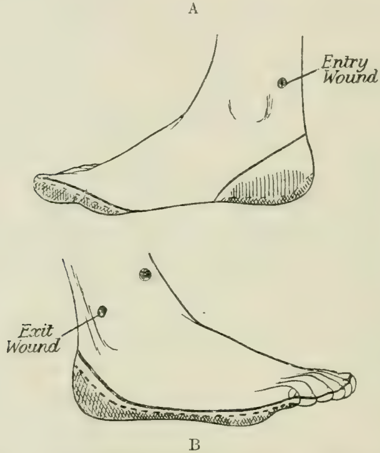

FIG. 5.—Changes in cutaneous sensibility in a case of incomplete interruption with irritation of the posterior tibial nerve (Case 3). The continuous lines in A and B represent the limits of loss of sensibility to cotton wool. The shaded portions are, in addition, insensitive to pin-prick. Over-reaction to pin-prick is represented by vertical lines.

In other cases, the symptoms and signs of irritation predominate, and the whole or greater part of the plantar area may exhibit intense over-reaction. Although voluntary movements are often retained in such cases, there is almost always some change in the electrical excitability of the muscles. The skin of the sole may appear smooth, glossy and pinkish in colour ; other trophic disturbances may be present in the form of profuse sweats or scaly desquamation. The actual lesion is often found to consist of slight adhesions between the nerve and the surrounding tissues, the freeing of which affords considerable relief, or the nerve exhibits fusiform enlargement—due to interstitial hæmorrhage—at the site of injury. The following example illustrates a case

in which the nerve was the seat of painful neuralgia, the whole plantar area showing over-reaction the freeing of adhesions greatly relieved the pain.

Case 4.—R. A., seen June, 1917, had been wounded through the calf by a bullet four weeks previously; for the past three weeks he has complained of intense pain in the sole of the foot, aggravated by any movement or upon the limb being touched, and which keeps him awake night after night; drugs have given little or no relief.

On examination, the small entry and exit wounds were seen respectively on each side of the junction of the middle and lower third of the shank. The whole of the plantar area exhibited marked over-reaction to painful stimuli. He flexed the toes somewhat weakly and reluctantly, stating that the movement gave rise to considerable pain. The long flexors of the toes responded normally to electrical stimulation, but he was unable to bear any attempt at testing the plantar and interosseous muscles.

The nerve was exposed ten days later by Major R. Jocelyn Swan and, in the track of the bullet, the nerve was found adherent to the tibialis posticus muscle posteriorly; there was also slight fusiform swelling of the nerve. It was freed from the adhesion and wrapped in cargile membrane.

When seen a month later, the patient still complained of slight pain in the sole of the foot (no doubt from the interstitial hæmorrhage), but stated that "it was nothing compared with what it was before"; the pain did not interfere with his sleep, and he was able to get about well.

Occasionally, a portion only of the plantar area exhibits over-reaction, the remaining being normal. In such cases it is almost invariably the internal plantar area that is affected, as in the following case:—

Case 5.—P. K., seen December, 1918, was wounded through the lower half of the calf by a shrapnel bullet in June, 1918. Ever since he has complained of pain in the sole of the foot; the pain experienced on putting the foot to the ground causes him to limp.

On examination, there was no actual sensory loss, but a portion of the internal plantar area showed diminished sensibility to cotton wool but marked over-reaction to pin-prick (fig. 4a). All movements at the ankle were normal and he was able to flex the toes, though performing the movement slowly and carefully. Flexor longus hallucis and longus digitorum reacted normally to faradism; the plantar muscles responded only sluggishly to faradism, and on no stop below No. 10 (2·0 microfarads) at 100 volts with the condenser.

It is probable that in this case the nerve was adherent on its inner side to the adjacent tissue (as in Case 6) and would have been greatly relieved by operation; unfortunately the patient was lost sight of.

The following case is of interest as it is one of the few that I have

seen in which causalgic symptoms were due to a localized adhesion of the nerve to its sheath and in which all pain was completely relieved by the freeing of the adhesion:—

Case 6.—P. W. was seen two months after having received a penetrating bullet wound through the calf. At the time of being hit, he had experienced acute pain in the foot, and for three days had complained of pain extending from the level of the wound down the back of the leg to the toes; following this the pain persisted in the sole of the foot only and would become much worse towards the end of the day. It was aggravated by any movement, cold and extreme heat, and the patient was reluctant to allow the foot to be touched.

On examination, there was marked over-reaction to pin-prick over the greater part of the internal plantar area and extending on to the inner margin of the foot; this area was somewhat diminished in sensibility to light touch (cotton wool). The external plantar and calcanean areas were normal (fig. 4, i and j). All movements at the ankle-joints were good but the patient was unable to flex the toes. Faradic excitability was diminished in the long flexors and scarcely present in flexor brevis digitorum. The former muscles reacted on No. 8 stop (0·5 microfarad) at 100 volts, but not below, with the condenser, while flexor brevis did not react below No. 9 (1·0 microfarad).

At operation, performed a few days later by Major R. Jocelyn Swan, the track of the bullet was traced towards the nerve, when it was found that about 1 inch above the level of the entry wound the nerve sheath was much thickened; on opening the sheath the nerve was found adherent on its inner side only; this adhesion was broken down. For about $\frac{3}{4}$ inch at this level the nerve appeared slightly more purplish and translucent than above and below. It was wrapped in cargile membrane and the wound closed.

The patient was seen about three weeks later and stated that all pain disappeared about fourteen days after the operation. There was no cutaneous over-reaction present and the toes showed good voluntary flexion. When re-examined a month later, beyond the presence of the scars, no abnormality could be detected.

Unfortunately, it cannot be claimed that all cases of causalgia yield so promptly to the liberation of the nerve from adhesions; not infrequently, no such adhesions are present, the nerve merely showing slight fusiform enlargement or a bluish and translucent zone at the site of injury. The intraneural injection of 90 per cent. alcohol or even section of the nerve above the level of injury may be necessary.

Of the sixty-four cases of posterior tibial nerve lesions, incomplete interruption had occurred in forty-eight. Of these, in thirty, flexor longus hallucis and flexor longus digitorum were involved, the injury in the remaining eighteen being below the origin of their branches of supply.

SUMMARY.

Injuries to the posterior tibial nerve are frequently overlooked, no doubt because they cause no striking paralysis, all movements at the ankle-joint being normal. More especially do they escape recognition when the lesion lies below the point of origin of the branches supplying flexor longus hallucis and flexor longus digitorum muscles, as in such instances power of flexing the toes is retained. In all wounds involving the calf where there is a complaint of pain or weakness in the foot, a systematic examination must be made of sensation in the plantar area and of the functions of the intrinsic muscles of the foot.

When the posterior tibial nerve is damaged, changes in cutaneous sensibility—varying from over-reaction to complete cutaneous anæsthesia according to the degree of injury—will be found over the sole, at the margins of the foot, and over the posterior and lateral aspects of the heel. There is also paresis and often atrophy of the plantar muscles and interossei, the toes being hyperextended at the metatarso-phalangeal joints and flexed at the interphalangeal joints. Flexion of the toes, and occasionally extension, may be less complete than in the opposite and normal limb.

When the site of injury is above or involves the branches of the posterior tibial nerve supplying the flexor longus hallucis and flexor longus digitorum muscles, flexion of the toes is either much diminished or is entirely absent. The ankle-jerk is also reduced or impossible to elicit. On faradic stimulation of the posterior tibial nerve on the inner side of the tendo Achillis, the response in the plantar muscles will be greatly diminished or absent. Similarly, stimulation of the motor points of the affected muscles shows reduced or absent reaction to faradism, a response only to the higher condenser stops—No. 8 (0·5 microfarad), to No. 12 (4·0 microfarads) at 100 volts, Lewis Jones scale—or, in complete lesions, no reaction even with the highest capacity, No. 12 (4·0 microfarads). The galvanic reactions vary with the degree of the interruption of the nerve functions.

When sensory disturbances of long standing are the predominant feature, exploration of the nerve should be considered. If the nerve shows complete interruption, the end-bulb of the upper segment should be removed or the fibrous cicatrix excised and end-to-end suture performed. In incomplete lesions exhibiting neuralgic symptoms the freeing of an adherent nerve will usually result in considerable relief.

Proceedings of the Section of Neurology of the Royal Society of Medicine.

Held December 9, 1920.

President—Dr. Ernest S. Reynolds.

Tabes: Its Early Recognition and Treatment.

By E. Farquhar Buzzard, M.D., F.R.C.P.

Dr. Buzzard first narrated the history of two patients suffering from tabes dorsalis. He then continued as follows:—

Tabetic Pains.

In order to justify a diagnosis of tabes in the absence of physical signs it is necessary to state one or two facts about which there can be little disagreement. In the first place, the disease is one which attacks the afferent paths of the nervous system, and it is only reasonable to suppose that its earlier manifestations are of a subjective character—in other words, that complaints indicating a disorder of function precede those which are clearly due to structural defects. In the second place, it will be agreed that in a large proportion of cases of unmistakable tabes careful inquiry will elicit a history, often going back five, ten, or more years, of pains which have been regarded as neuritic or rheumatic in origin, and have not been associated with any other disability. Not once but a score of times have I asked a tabetic patient whether he has suffered from pains. After repeated denials I have altered the wording of my question and inquired for rheumatism. "Oh, yes, I have suffered from rheumatism for years." "What do you mean by rheumatism?" "Why, sharp, shooting pains in my limbs." This conversation shows how little the patient has associated his pain with the disability for which he is seeking advice, and incidentally how few patients understand the language they speak.

Granted that pains can be, and often are, the earliest signs of tabes, can they be distinguished from pains of other origin? I would answer this question in the affirmative and even go so far as to say that the pains of tabes described by a patient who is able to translate his feelings into intelligible language are quite pathognomonic. The fact that they are called lightning pains and that patients often say that they shoot up and down their legs has been responsible for a very general misunderstanding of their true character. When the patient states that they shoot up and down the limb he does not mean that each pain shoots from the hip to the foot and vice versa. He means that during an attack he may have pains first in the thigh and a few minutes later in the foot. If he is asked to describe them in greater detail he will inform you by gesture and by word that these pains have an axis, so to speak, at right angles to the surface of the limb. They stab like a knife or a darning-needle going in, or they resemble the effect produced by taking up the flesh, pulling at it, and letting it go again. Further, the pains come not singly, but if not in battalions at any rate in platoons. They are rapidly repeated, several occurring in the course of a second or two, followed by a lull of longer or shorter duration. Bouts of paroxysms may last for a few hours to a few days and then cease for a time. Favourite sites for these pains are the heel, the inner aspect of the shin, and the inner aspect of the knee. The area within which they repeat themselves is small, described by the patient as the size of a crown-piece, or no bigger than the palm of the hand. Between the frequent paroxysms these areas are often tender to light touch, so that the patient does not even like the contact of his clothes. Many patients are quite certain that pains may be provoked by a cold draught, by a change of weather, or even by a glass or two of beer. Others are equally sure that the responsibility lies with fatigue or anxiety. In the early stages of the disease pain rarely occurs during active exercise, but later on walking may be brought to a full stop by a sudden and overpowering paroxysm.

Before leaving the subject of pains let me make it quite clear that those I have described are not the only ones suffered by tabetic patients. There are deep, gnawing, aching pains in the bones, and others which have not the same diagnostic value. Let me also emphasize the fact that tabetic lightning pains must be recognized by their characters and not by their severity. Many patients confess to what they call "niggling pains," trivial in respect to suffering, but identical in nature with the lightning type.

It would be surprising if there were not other subjective phenomena in early tabes, and attention may be drawn to the not uncommon complaint that hot or cold water has become disagreeable or unbearable, that the patient is obliged to have his bath at a temperature which is described as tepid. Not that the hot or cold water provokes pains, but that it gives rise to a sense of great discomfort. This often applies to the whole surface of the body, although pains may be absent or confined to the lower limbs. Similarly it is not uncommon to hear that the fingers are numb when it is quite impossible to determine any disorder of sensibility by tests. This, of course, is not pathognomonic of tabes.

Treatment.

The necessity for advising antisyphilitic treatment of tabetic patients is not now so urgent as it was a few years ago, but it may be doubted whether the principles of this treatment are yet sufficiently recognized. We are all asked: "How long must I go on with treatment before I am cured?" For many years my answer has invariably been, "For the rest of your life." I am never consulted about a primary chancre, but if I were my advice would be the same. It cannot be denied that we are not and never have been in a position to tell a patient that he has been cured of syphilis, and, as neurologists, we are now constantly seeing tabetic patients who have been cured of syphilis by the arsenical compounds, just as fifteen years ago we saw those who had been cured by mercury. I maintain that the only honest advice we, as medical men, can give to patients suffering from any syphilitic disease is to the effect that they should continue to have periodic courses of treatment for the rest of their lives. Tell them that prevention is better than cure, and that they must regard this precaution as a method of insurance. In my experience so many victims of tabes and other syphilitic nervous diseases have allowed valuable time to slip by owing to negative Wassermann reactions that my confidence in this test as a method of regulating treatment is shattered.

Conclusion.

Is there any lesson to be learned from the story of the two patients which I have presented to you? The first man was infected with syphilis fifteen years ago, and has undoubtedly suffered from tabes for twelve years, during eight of which he has been under treatment. To-day he would be passed as a first-class life by any medical officer of an insurance office who did not know his history. It would be unwise

and unjustifiable to say that he is cured, but it is difficult to believe that the natural progress of his disease has not been arrested. And it must be remembered that he wasted four years before his symptoms were recognized and treated. The second patient was more fortunate. Only six months were wasted, and the result of treatment was rapid and lasting, judged by the fact that he has now enjoyed six years of freedom from any kind of symptom. These are not the only cases of the kind which have come under my observation, but I regret to say that it is a rare experience to be asked to see a patient suffering from tabes whose symptoms have not been of long-standing.

It is obvious that the value of these observations depends on the reliance which can be placed on the description of pains given by patients, and on the criterion the description affords for purposes of diagnosis. In many cases the description is vivid and easily recognized. In others it can only be elicited by patient cross-examination, avoiding the employment of leading questions. After many years of critical study of this problem I am convinced that the lightning pains of tabes are, for practical purposes, pathognomonic, and that they can and should be recognized before they are many months old. Treatment initiated before physical signs are apparent is not only possible in many cases, but is likely to be attended by good results if persevered with to the end of the chapter.

The Effects produced by obscuring the Vision of Pigeons previously deprived of the Otic Labyrinth.

By SYDNEY SCOTT, F.R.C.S.

Two pigeons were shown, in whom successful operations had been performed, and Mr. Scott made the following remarks:—

The research was originally undertaken to ascertain whether pigeons whose labyrinths had been destroyed would in the course of time regain power of flight, which is temporarily lost by interfering with the labyrinthine functions. With permission of the Home Office the experiments were commenced in the autumn of 1919. Under general anæsthesia absolute alcohol was injected into the labyrinth with a hypodermic needle, which was passed through the tympanic membrane and made to penetrate the base of the columella into the vestibule. This procedure was carried out with the aid of a small

aural speculum and bright head light, as employed in the operation of incising the tympanic membrane in cases of otitis media in human beings, smaller instruments, of course, being required. In some pigeons only one labyrinth was destroyed, in others both were destroyed, either immediately or after an interval of some days. In the course of nine months or thereabouts, thirteen pigeons were operated upon. Several difficulties were encountered in the first series before it was found possible to keep the birds alive for sufficiently long periods to enable me to make observations on their powers of flight. One died from the anæsthetic alone. Another collapsed under chloroform, but recovered after inversion and artificial respiration. In another bird the base of the columella could not be felt and the operation failed on three separate occasions to produce any sign of labyrinth destruction, such as forced movements, with inco-ordination, and disturbance of gait, flight, or assumption of abnormal posture. Another bird died instantly at the moment of injection; too deep penetration and excessive quantity of alcohol injected would account for this sudden effect on the brain. Among two survivors, it was evident that the alcohol had acted on one labyrinth only in each case, though both ears had been injected. In these two birds, on recovery from the anæsthetic, the forced movements and altered behaviour was of such a character that it was evident that the right labyrinth was rendered functionless in one bird, and the left labyrinth in the other. Of those which survived the actual operation, four died between the seventh and the eleventh days, apparently of faulty nutrition, though hand-fed after the operation. On this account it was decided to keep the birds for two months before operation to accustom them to be hand-fed daily during that interval, and afterwards as long as was found necessary. Eventually two successes were obtained, both birds surviving over nine months, during which time numerous observations were made on their behaviour and powers of flight, one with only one labyrinth destroyed, and in the other with both labyrinths successfully destroyed.

In order to preserve permanent records of the movements and behaviour of these birds about 1,400 feet of cinematograph film pictures were obtained at various intervals. These films were demonstrated.

Behaviour of Pigeons both Labyrinths of which were destroyed.

July 17, 1920.—10.8 a.m.: Chloroformed. 10.10 a.m.: Absolute alcohol injected into left labyrinth. 10.12 a.m.: Absolute alcohol injected into the right labyrinth. 10.20 a.m.: Recovering from anæs-

thetic; tendency to hold head back, and when standing to fall directly backwards; irregular head nystagmus; no ocular nystagmus observable.

July 21, 1920.—Quite active. Tries to peck grain-food, but tends to pitch forwards when beak touches floor level. Appears to be trying to avoid pecking too far forwards. Misses grain by about a centimetre, eight to ten times, before picking up. Stands with wide base, but not asymmetrically. Within two months this bird walked about and could fly up to perch 2 ft. high. Marked tendency to overbalance backwards, always holding beak high in the air. When removed from cage flew straight in a room, but misjudged when landing, and pitched forwards on to beak. Wing movements symmetrical; the bird did not fly round in a circle, or appear eager to fly at all when released occasionally.

Effect of covering one or both Eyes of the same bird with a Hood.

When both eyes are covered with a hood, the bird remains motionless in whatever position it is placed. If standing, when eyes are covered the beak goes forwards, rests on the floor, or the bird overbalances backwards, turning a somersault till the head rests with neck flexed beneath its back on the floor. If the eyes be covered and the bird be laid out like a dead bird, it will remain motionless except for respiratory movement an indefinite time. It was found to behave in the same manner when in a dark room, the observer judging the bird's posture by feeling with the hand. When in the light—only one eye was covered—the bird walked around slowly in a circle, the centre of which was towards the open eye.

About six months after the injection this bird could fly from the floor up to a window, and though it never landed easily like a normal bird, it learnt how to prevent itself pitching forwards violently on to its head. It never learnt to peck accurately at grain, and though it attempted to feed itself early, and hand feeding became unnecessary in a few weeks, still it was found advisable to supply it with a cup of grain instead of with grain scattered on the floor. It evidently experienced difficulty in selecting its food.

Incidentally the bird appeared to be deaf, that is to say, it never showed the movements associated with startling noises, such as explosion of old electric light bulbs, as its normal or unilaterally defunct companions did. Moreover the movements of the head were more rapid and constant as if the bird were dependent solely upon vision, which it probably was. When hopping from ground to perch the

balancing on the perch was at first accompanied by swaying movements in an antero-posterior plane, the bird sometimes falling backwards (never forwards) off the perch.

Unilateral Destruction of the Labyrinth.

In marked contrast with normal birds and the birds with both labyrinths destroyed, the behaviour of those with only one labyrinth destroyed is markedly different and quite characteristic.

The following is an example: A blue chequer pigeon was injected in July, 1920, and survives in May, 1921.

July 17, 1920.—11.16 a.m.: Chloroformed. 11.20: Absolute alcohol injected into left labyrinth. 11.30: Recovering from anæsthetic and runs or flutters circus-wise to the left.

July 28, 1920.—Stands erect until disturbed; when attempts are made to handle the bird in its cage, it tries to evade the hand and flutters round to the left and drops the left side of the head to such an extent that the head is axially rotated until the lower beak becomes uppermost, and the bird holds its head with the vertex resting on the floor of the cage. To a less extent it is obvious that the left side of the body is also lower than the right. The bird remains still in this position, looking with its right eye at an observer who is standing on the bird's left side. In a minute or so the bird suddenly resumes its normal posture and stands erect. When removed from the cage the bird may walk straight a little way, but if pursued it spreads its wings and flutters to the left, eventually reaching some corner which arrests progress and there re-assumes the position of axial rotation of the head as described above. If the bird be held in the hand off the ground and coaxed to fly, it invariably goes in a circle, alighting on the ground, and shows no desire to resume flight. The bird never flies or hops up to its perch like the bird with both labyrinths destroyed. It sleeps on the floor of the cage.

One bird whose labyrinth had been destroyed on one side attempted to fly when released on the roof of a building, but on reaching the parapet its flight movements were definitely circus-wise, and it lost height, resembling the "spinning nose dive" of an aeroplane, landing in a heap on the ground 72 feet below. The bird appeared to be dazed but was not apparently injured, and lived for some few weeks afterwards. However, it was decided not to allow others to escape and possibly injure themselves in this way.

The Effect of obscuring the Vision in Pigeons devoid of one Labyrinth.

When a hood is placed on the head and both eyes are covered, the bird keeps still, with the head axially rotated to an extreme degree as previously described. When only the left eyeball is uncovered (assuming the left labyrinth is destroyed) the bird tends to hold the head in the normal erect position and walks slowly in a circle towards the left. When only the right eye is uncovered (the left labyrinth having been destroyed) the bird reassumes the axial rotation of the head with the left side downwards and the underbeak uppermost, while it excitedly executes rotatory movements to the left. Normal pigeons when hooded will walk about to right or to left or forwards, or stand on a perch, or sit at roost, but will not remain passive or overbalance themselves like hooded birds which have been deprived of one or both labyrinths.

CLINICAL MEETING HELD AT THE NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC, QUEEN SQUARE, FEBRUARY 10, 1921.

President—Dr. Ernest Reynolds.

(1) *A Case for Diagnosis; possibly Tabes Dorsalis.*

By Dr. Tooth.

M. D., female, aged 35.

Family history *nil ad rem.*

Ten years ago transient attacks of loss of vision, which recurred several times during the next four years ; none since then.

Five or six years ago began to have trouble in walking, tendency to walk quickly and fall forwards ; in hospital in 1917 for ten weeks ; improved for about twelve months, then trouble began to recur and has gradually got worse since.

Twelve months, some hesitancy in micturition.

Six months, girdle pain on the right side.

Twelve months ago legs gave way and she had complete right hemianæsthesia and hemianalgesia for a fortnight.

At Christmas, 1920, had anæsthesia and analgesia left lower limb, lasting six weeks.

Present state.—Optic discs physiologically pale ; pupils react to light sluggishly ; nystagmus in horizontal direction on extreme lateral movement,

especially to the right and in vertical direction on extreme upward movement. Sensibility to pin-pricks slightly diminished over right half of body and slightly less keen than normal all over. Deep muscle-pain sense in legs absent. Touch: localization of touch, passive movement, stereognosis good. Power slightly diminished in legs, and legs hypotonic. Co-ordination fairly good, slight tremor in hands on finger-nose test. Arm jerks, knee and ankle jerks absent; right abdominals present, left absent; right plantar slight flexor, left extensor. Gait unsteady. Rombergism present. Wassermann in blood and cerebrospinal fluid negative.

(2) *A Case of Acute Myelitis at about Seventh Dorsal Segment, with Optic Neuritis.*

For Dr. TAYLOR, by Dr. SHEPHERD.

G. S., female, aged 23. Woke up on July 15, 1920, with pain over the left eye; the sight went in that eye by the next morning and she had pains in the legs and back during the night. She was feverish and had headache the next day and the right eye became similarly affected, so that by the next day she was absolutely blind and was completely paralysed in both legs with analgesia and anæsthesia up to the waist, and retention of urine and fæces.

Legs were quite spastic for ten days and then the spasticity gradually went off and the legs became quite flaccid. Eyesight gradually improved, so that she can see quite well now. From October, 1920, incontinence of urine.

Physical examination.—Optic discs pale, especially the left, showing evidence of old optic neuritis. Complete paralysis of legs below waist (very slight flicker in toes only). Complete loss of sensation to cotton wool, pin-prick, temperature, from below the waist; the loss to pin-prick extends an inch higher than the loss to cotton wool, and in the area between these two upper levels hot and cold is wrongly appreciated. Arm jerks normal, knee and ankle jerks, abdominals and plantars absent.

Cerebrospinal fluid: albumin 0·6 per cent., N.A. strongly positive, no cells seen, Wassermann negative. Wassermann in blood negative.

(3) *Case of Intracranial Pressure (?) Tumour; Recovery without Operation.*

For Dr. TAYLOR, by Dr. SHEPHERD.

E. F., female, aged 28. At the age of 16 suffered with headache and loss of power in the hands and legs, especially the right side. Headaches became very severe and continuous and were accompanied by vomiting. She was admitted to the hospital in October, 1904. She had then marked papillœdema, but the headache and vomiting subsided soon after admission and the power gradually returned to the limbs.

About three months after admission she was able to be up and walking, but the arms and legs got weak again: she was very unsteady and had to go back to bed, so that she was still very weak about August, 1905. She gradually improved again and was able to be about in six months.

From that time until eighteen months ago she had been fairly well, with the exception of some tremulousness and weakness in the legs, especially in the right.

During the last eighteen months the right leg has become weaker.

No visual or sphincter troubles; only a very occasional headache; some tingling in the hands and feet during the last few months.

Optic discs normal; (?) slight nystagmoid jerking on looking to the right; otherwise cranial nerves normal. Movements of right leg much diminished with slight talipes equinus on right side. Abdominal reflexes active and equal, knee jerks and ankle jerks active and equal; clonus, right plantar inactive, left flexor. Walks dragging right leg.

(4) *Case of Friedreich's Ataxia.*

For Dr. TAYLOR, by Dr. SHEPHERD.

F. L., male, aged 19.

No family history. Patient has one sister and one half-brother.

Patient began to be unsteady in walking at age of 7 years; says he always fell upon his nose; this gradually became worse, legs weak and muscles gradually wasted, so that by age of 14 he was confined to a chair.

No change during the next three years, but has been feeling stronger during last two years. Deformity of the spine, said to have developed during last three years. Pes cavus present since childhood. No alteration noticed in speech.

Patient is rather facile boy of 19, not keen mentally, bodily nutrition bad, arms wasted. Nystagmus present, especially to right. Also slight shaking of head; speech slow and of scanning variety; motor power diminished in lower extremities and in trunk, with wasting in legs; no spasticity. Slight pes cavus, slight intention tremor. Deep reflexes absent; plantars extensor. Slight kyphoscoliosis. Wassermann negative in blood and cerebrospinal fluid.

(5) *A Case of Lethargic Encephalitis.*

By Dr. JAMES COLLIER.

Mary B., aged 20, was seized with intermittent frontal headache on December 26, 1920. On the following day her eyes became crossed, and she saw double and became very sleepy and ceased to talk or to move spontaneously. She had considerable trouble with retention of urine. She was not laid up, and attended her doctor regularly. She has remained in this condition since, and during this time her memory has been good; she has obeyed orders promptly and replied to all questions promptly and correctly. Her facial and bodily expression is one of complete statuesque immobility, in lying, sitting, or standing. She will remain in any awkward attitude in which she is placed for long periods. She never moves spontaneously except in response to the more urgent calls of nature. She does not turn over in bed. She presents no emotional expression, positive or negative. She never speaks

spontaneously. She obeys every word of command at once and correctly. She answers every question at once and correctly, and gives a correct and detailed account of her illness if she is catechized. In response to the usual salutation she answers always: "I feel quite well, thank you."

There is severe bilateral ptosis which is diminishing. Complete internal ophthalmoplegia with large pupils. The axes diverge in vertical parallelism. Downward movement absent. Upward movement almost absent with quivering. Lateral movements of about two millimetres with quivering. Vision very poor, but no change in media or fundus.

No other brain-stem paralysis.

On admission to St. George's Hospital, January 12, 1921, the left abdominal reflex was diminished and the left plantar was extensor. Now the abdominal reflexes are equal and the plantars are flexor. Some fibrillation was noticed in the right anterior tibial and peroneal muscles.

Under observation she has developed dropped toes and dropped in-turned foot on the right side with severe weakness of the external sciatic group of muscles, with increased tendon jerks.

The points of interest are the peculiar clear but immobile mental state and the condition of the right foot.

(6) *Case of Amyotonia Congenita.*

Shown by Dr. COLLIER.

V. C., female, aged $4\frac{1}{2}$.

Child born normally. Difficulty in sitting up; has never walked, and only began to crawl ten months ago. Pain in foot on being stood up. Otherwise no complaint; no fits nor injuries. Bright child, teeth and talking at normal age. Ten months ago had headache, vomiting and screaming for three days; otherwise no illnesses. Clean in habits. Father died of consumption. Three siblings dead—one of meningitis, at 5 years; two at 4 and 3 months, having been sickly from birth: one alive, aged 13, and healthy.

Child of poor development, good intelligence, and unusually good-tempered and uncomplaining. Stands with support, hips and knees being flexed from contractures of muscles, and feet slightly plantar-flexed. Muscles of lower limbs only of poor tone and somewhat wasted, but moderate power. Bears stretching of contracted muscles well, and has improved considerably since admission. Knee and ankle-jerks present. Abdominal reflexes present. Plantars flexor.

(7) *Case of Graves' Disease; (?) Paralysis Agitans.*

Shown by Dr. COLLIER.

M. B., female, aged 26.

Lump on throat noticed eight years ago; has got smaller, if anything, during past six months. Palpitations and shaking for six months. Perspiring for two months. Menses regular—scanty. No exophthalmos.

Fine tremor of hands. Coarse rhythmical tremor of feet, stopping for a time by voluntary effort and then beginning with renewed force; lateral as well as up and down movement. Sometimes general tremor of body; some of tongue.

Attitude and mask-like face resembling paralysis agitans. Reflexes normal.

(8) *Case of Encephalitis Lethargica.*

By Dr. BUZZARD.

B. D., aged 43.

Family history.—Father died of carcinoma of liver: brother has neuritis in his arms. Previous history: good, but patient "nervous."

September, 1920: Pain in both legs, which makes walking difficult, but soon improved.

November, 1920: Pain in right knee and weakness of right ankle; no numbness, but foot gets cold. No complaint of arms. Right foot "dropped" three weeks.

January 5, 1920.—Examination: Fibrillation of deltoids, pectorals, and triceps; more on left side. Weakness of sterno-mastoids and of above muscles. Slight weakness of extensors of wrists. Fibrillation of right quadriceps and wasting of all muscles of right leg: foot dropped. Some weakness of left leg; much weakness of right, especially of extension of foot and toes. No incoordination.

Sensation: Pain in right calf; deep pressure of muscles of legs painful; some loss of pin-pricks and heat and cold inner side right leg.

Reflexes: Arm and knee jerks increased, right ankle jerk absent, left present, no clonus. Abdominals brisk. Plantars: right flexor, left extensor.

(9) *A Case of Echinococcal Cyst of Left Parietal Region.*

By Mr. SARGENT.

M. E., aged 15.

April, 1920: Weakness of right hand, progressing to whole arm.

July, 1920: Peculiar feeling in right arm and face, starting in fingers and followed by vomiting.

September, 1920: Similar attack.

October, 1920: Right foot caught in walking.

December, 1920: Optic neuritis noted.

December 25, 1920: Pain in left frontal region; noises in ears; drowsy; vomiting; some defect of speech.

January 2: Slight hesitancy of micturition.

On admission into hospital, January 4, vision $\frac{5}{5}$, papillœdema, R. + 6D., L. + 5D. Right lower face weak, right hemiparesis arm and leg, slight loss of sense of position, localization of touch, and recognition of objects on right limbs. Pin, cotton wool, and vibration less distinct on right. Reflexes: deep, increased on right, abdominals less active on right; plantars, right extensor, left flexor.

January 14, operation: large cyst removed reaching surface just behind left fissure of Rolando, contained 60 c.c. of fluid, diameter about 6 cm. Pathologist's report: echinococcal cyst; fluid, albumin 0·015 per cent., sugar a trace; chlorides 0·57 per cent., sp. grav. 1·006.

After operation a few fits with twitching of right face, tasting movements of lips. Rapid general improvement. Subsidence of papillœdema.

(10) *Case of Spinal Tumour removed by Operation.*

By Mr. SARGENT.

A. W., aged 42.

June, 1918: Pain left side round iliac-crest.

June, 1920: Operation on left kidney, pain slightly improved, but soon recurred.

Three weeks later numbness of both legs, left foot dropped, pain reached left foot; soon right, also weak and painful. Slight hesitancy of micturition.

Examination: Spine not tender, but sitting up painful. Spasticity of both legs; some wasting; fibrillation of hamstrings and calf muscles. Spastic and unsteady gait, Reflexes: knee jerk very brisk: ankle jerk present. Plantars: double extensor. Abdominals absent below umbilicus. Sensation: pin-prick lost; L. 2 – S. 2 R.; D. 9 – S. 2 L.; sense of passive movement in toes lost. Cerebro-spinal fluid: yellowish fluid coagulating solid in tube; cells absent; albumin 3·70 per cent.; Wassermann reaction negative.

Operation, November 11, 1920: Tumour seen and felt through dura opposite cut arches of D. 11 and theca opened without wounding arachnoid; tumour sub-arachnoid, soft and encapsuled on posterior and left lateral aspect of cord; removed and theca sutured.

Pathologist's report: Myxomatous tumour with clumps of endothelial cells.

(11) *Case of Optic Atrophy from Neuritis and Gastric Attacks.*

By Dr. WILSON.

W. C., aged 56.

Family history: Father died of cancer.

Past history: Syphilis at 24.

1898: Sight impaired, treated at Moorfields in 1901 with mercury.

1902: Pain in stomach two hours after food, relieved by vomiting, improved since 1915 until recently. No unsteadiness of walking and no sphincter disturbance.

Present state: Visual acuity: right, $\frac{1}{50}$; left, $\frac{1}{30}$; on looking to right of fixation point, at that point vision almost nil.

Fields: Loss of upper half and general constriction, with central scotoma.

Fundi: Old atrophy. Argyll Robertson pupils. No ataxy. Deep pressure of calves very painful. Deep reflexes absent. X-ray suggests cicatrix from old ulcer or growth of stomach with delay in emptying of stomach. Wassermann reaction: negative in blood. Cerebrospinal fluid: no increase

of cells, globulin in cerebrospinal fluid. Blood count: red cells 3,700,000; hb. 70 per cent.; white cells, 10,500.

(12) *Case of Paralysis Agitans following Malaria.*

Shown by Dr. S. A. K. WILSON.

H. C., male, aged 35.

Malaria in England in 1918, blood tested at special malaria hospital (Connaught Hospital). A month later had "kicking" and twitching movements of left side, which improved after May, 1919.

In December, 1919, had weakness of left side and some slight stiffness of both hands. He began to stoop and to have difficulty in turning over in bed. By May, 1920, his right leg was also stiff. August, 1920: tremor, chiefly of right hand, slowness of speech and difficulty in opening mouth.

Examination: Slight weakness of left lower face. Spasm and rigidity of sterno-mastoids and trapezii. Tongue deviates to right. Mask-like face. Rigidity of trunk and proximal limb muscles. Pill-rolling tremor of hands. Paralysis agitans attitude and gait, with festination and retropulsion.

(13) *A Case of Epilepsy with Acromegaly and Unilateral Tremor.*

By Dr. J. TYLOR FOX.

W. G. O., born November 27, 1895. Nothing neuropathic recorded in the family. This patient's history and present condition may be classified under three headings; his epilepsy, his unilateral tremor, and his dyspituitarism.

Epilepsy.—First fit at fifteen months, second three or four years later; after that about every three months. Since admission to Lingfield Colony eight and a half years ago, he has had attacks of petit-mal every two or three months with epigastric aura, loss of consciousness without spasm lasting two or three seconds in all, also three major fits in the last two years.

Neurological signs.—Has had tremor of the left arm and leg "as long as he can remember." The tremor is continuous, apparently unaffected by voluntary movement, coarse, averaging five or six a second, and most marked in, if not confined to, the muscles about the large proximal joints, i.e., the shoulder, hip, knee and elbow. It ceases during sleep.

The left upper arm is $\frac{3}{4}$ inch less in circumference than the right. The forearms, thighs and calves appear to be equal on the two sides.

Reflexes, superficial and deep, give a normal response, though those on the left side are rather slower than on the right. Dynamometer readings 110 each side. No vasomotor differences between left and right side. No interference with sensation. No tremors of face, but coarse tremors of tongue. Optic discs, normal. Field of vision by rough perimetric tests, normal.

General signs.—Curvature of spine said to have been first noticed at fifteen months and attributed to rickets. Teething is also said to have started at fifteen months, walking and talking at eighteen months. Skeletal changes most

marked in skull and spinal column. Cranium tilted backwards on the facial portion of the skull. Superior and inferior maxillæ, large, especially inferior. Teeth pitted and grooved, and in upper jaw separated from each other. Nostrils, large and broad. Supra-orbital ridges, well developed. Eyelids, thick. Tongue, large. Well-marked kyphosis with scoliosis to the right in dorsal region. Lordosis in the lumbar region. The skiagram taken at the National Hospital, Queen Square, of the skull of this patient quite negative. The bones of the skull are normal in appearance, the sella turcica is of about normal size, its outlines can be well seen; the posterior clinoid processes are quite distinct, and the cavity does not encroach on any of the fossæ in front of it at all. As far as the skiagram goes, there is nothing abnormal to be made out. Right testis undescended and removed some years ago. Left testis very small. Thyroid not palpable. "Has never had a headache in his life." Temperature 97° to 98° F. Systolic blood-pressure, 110. Urine, daily quantity 49 oz., no sugar, no albumin. No diminished sugar tolerance. Patient is of fair intelligence, but his memory is poor.

(14) *Case of Torsion Dystonia.*

By Dr. BLANDY (for Dr. COLLIER).

H. R., aged 8, male.

History.—Six months after an attack of diphtheria (aged 5) involuntary tonico-clonic movements of an extensor type developed, with in addition perverse voluntary movements of the hands. The condition has shown marked remissions, but with each exacerbation has become more intense and more extensive.

First noticed in the right arm, with "loss of use of the hands," the movements gradually involved the neck, face, tongue, then left arm, trunk, and finally, in the severest stage, the legs.

February 1920, after being knocked down by a bicycle, there was a history suggestive of a tonic fit, followed by an exacerbation of extreme degree, associated with difficulty in eating, drinking and sleeping, and in starting the organic reflexes, owing to the excessive movements.

He could only sleep on his face, head extended, right arm tucked in behind his back.

Past history.—Nothing abnormal till aged 5, since when has stopped growing, and has had an intermittent morbilliform rash.

Family history.—Father has "bronchial asthma." Sister had chorea after rheumatic fever, maternal grandmother, mother, aunt, two uncles, had enlarged thyroids (the aunt died of Graves' disease).

Physical signs.—Small for age, intelligent; normal cranial nerves, sensation, reflexes. Wassermann reaction negative in serum and cerebrospinal fluid.

Motor system.—Hypertrophy of muscles most in spasm. Hypotonia during relaxation—no tremor nor ataxia, good power. Voluntary fine movements of hands awkward and slow. Hands in posture of flattened fists. Intermittent

clonico-tonic, tic-like, bizarre, involuntary movements, i.e., extension of head and shoulders with protrusion of tongue, &c. Extension of right arm with over-pronation of wrist, &c.

The child is at present date in a remission.

(16) *Cholesteatoma in Ponto-cerebellar Angle.*

Shown by Dr. J. G. GREENFIELD. (Dr. RUSSELL.)

Henry M., aged 27.

History.—Staggering gait, six months; left-sided deafness and headache—one month. Handwriting shaky, and difficulty in swallowing, a few weeks.

On examination: Papillœdema right and left. Coarse nystagmus to left, fine to right. Progressive nerve deafness on left side. All deep reflexes over-active. Double ankle clonus. Plantars upwards (Babinski) right and left.

Post-mortem examination three months after admission: Large cholesteatoma of left ponto-cerebellar angle, passing over the side of the pons under the basilar artery. Eighth nerve flattened, and internal auditory meatus enlarged and containing a prolongation of the tumour.

(17) *Colloid Ball-like Tumour in Third Ventricle, immediately over the Infundibulum.*

Shown by Dr. J. G. GREENFIELD. (Dr. RUSSELL.)

John G. C., aged 36.

History.—Headaches, worse when tired, sixteen months; diplopia, two weeks before admission. No vomiting.

On examination: slight papillœdema right and left; diplopia on looking to extreme left, from paresis of left external rectus. Left facial paresis.

Motor and sensory systems normal except for coarse tremor of outstretched hands.

Reflexes: all normal.

One week after admission he had several "fits" in which he rubbed the back of his head and was "queer" in his manner. He remembered nothing of them afterwards.

Post-mortem examination two weeks after admission: Ball-like encapsuled tumour in third ventricle causing hydrocephalus of both lateral ventricles. It is extremely tough after hardening in formalin, and feels like a solid rubber ball. Microscopically it is composed of colloid with a fibrous tissue capsule.

RECENT THEORIES OF HEARING.

The Analytical Mechanism of the Internal Ear. By Sir THOMAS WRIGHTSON, Bt., D.L. 254 pages + ix. London: Macmillan, 1918.

Under the above title Wrightson describes the mechanism of the sense of hearing, from the reception of the sound waves by the drum, to their arrival as auditory sensations at the brain.

The subject matter may be considered in the following separate sections;—

(1) The physical properties of sound.

(2) The ossicles and ear drum.

(3) The cochlea and its contents.

(4) The hypotheses of audition.

(5) Appendix by Keith on the anatomy of the ear.

Section 1.—The first part of this section, which is introductory in character, deals with the physical properties of sound, the formation of beats, summation and difference in tones and the like. The description is interesting, simply put, and does not call for special comment.

The ohmograph, of which a full description is given, does not appear to be novel except for a few mechanical details. The mathematical analysis of certain compounded sine curves which follows must now be considered in detail, since it forms the basis of the hypothesis of audition which Wrightson states later in his book. The process of analysis which Wrightson described may be briefly stated as follows: Three curves are prepared, the sine curve of a note A, the sine curve of another note C, and the curve obtained by summing both of them by the ohmograph. A pair of callipers is now taken, set to the wave length of the note A, and then tried on each crest, trough and crossing point of the compound curve in turn; if the two feet of the callipers fit any of the distances on the compound curve, such distances are marked A1 and A1. The next pair of points found to correspond to the distance between the feet of the callipers is marked A2 and A2 and this process is repeated until one complete cycle has been examined. In one case three such distances were found, and in another four. If now the whole process be repeated with the wave length of the note C, then again a number of distances between crests, troughs and crossing points in the compound curve are found, which correspond with the wave length of the note C. In one case five such crossing points were found and in another three. If finally the above process be repeated with a curve compounded of three or four different wave length sine curves,

then as before a number of distances are found on the compound curve, which are equal to those on each of the constituent curves.

Wrightson now goes on to show that if the wave length of the summation and difference tones be taken, then as before distances equal to these are found on the compounded curve. Wrightson draws the conclusion, therefore, that when two or more musical notes are sounded together, the compound curve contains a number of "impulse points," the time intervals between certain of which represent those occurring in both the constituent musical notes and also in their summation and different tones. Wrightson points out later that it is the recognition of these time intervals by the brain which enables us to recognize these different constituent tones.

Wrightson now goes on to state (p. 30) that no mathematical exactness is postulated between the time periods in the compound sound waves and those of its constituents, yet they may be sufficiently accurate for the theory, "as the ear, in judging of musical intervals, does not depend upon absolute accuracy of vibration ratio, a fact well known to practical musicians." This want of exactness is readily detected in the diagrams which Wrightson gives; thus, taking the first (fig. 3) as an example, it will be found that the distance A_2-A_2 is smaller than the wave length of A, while A_3-A_3 is larger.

Boring and Titchener[1] have pointed out that taking the above analysis at its face value the following criticisms can be advanced against it. (*a*) The summation and difference tones are both an octave too low. (*b*) Besides the pure tone the ear should also hear according to Wrightson the octave below it; this the ear certainly does not do. (*c*) It should also hear the octave above it, and this again is not the case. The reader will conclude from this that even if the fact is ignored that the mathematical analysis is not exact, it still further prejudices itself by leading to false deductions. The proof must now be given that it is the analysis itself which is at fault; supposing that the compound curve in fig. 3 be again turned to and that the callipers be set not to the wave length of either A or C, but to a purely arbitrary distance somewhere between these two values. Then if this distance be laid off as described above, it will probably be found that it is several times repeated in one cycle. I performed the process for twenty arbitrary distances and found each of these distances represented some three or four times in the compounded curve. This at first sight astonishing result was partly explained by finding that the total number of distances in one cycle varying between 1·5 mm. and 44 mm. in length was approximately 400. Assuming that all distances are equally represented, each distance would vary from the next by approximately 0·1 mm. If we assume an inaccuracy of setting of say 0·1 mm. then any arbitrary distance between 1·5 mm. and 44 mm. will be represented on the average three times (one exact fit, one 0·1 mm. too large, and 0·1 mm. to small).

We thus see the explanation of Boring and Titchener's remarks. "It is strange" they say, "that the theory, if we take the diagrams at their face

[1] *American Journal of Psychology*, April, 1920, vol. xxxi, pp. 101-113.

value, should thus insist on proving itself. Moreover, there is something disconcerting about a theory that will come right on any assumption." This mathematical foundation that Wrightson had laid for his theory therefore fails since the brain cannot be expected to pick out certain time intervals, and construe them as musical notes when apparently all time intervals between certain limits are equally represented,

Section 2 contains a well illustrated description of the ossicles, their suspensions and musculature and the way in which they function. Wrightson now attempts to calculate on "static" principles the total increase of effective pressure due to the lengths of the ossicular levers and the areas of the drum and the piston of the stapes. It is clear to the reader, however, that the calculation should be a "dynamic" one, since the levers when functioning are in rapid motion. For not only must work be performed in setting and keeping the ossicles in vibration, but also in overcoming friction at the joints between them, and the damping action of the air surrounding them.

Section 3 contains a description of the cochlea and of the organ of Corti. The anatomy and histology of the parts concerned is well described, and easily understood. When, however, Wrightson commences to describe the calibration of the cochlea the argument becomes very difficult to follow; for the ideas are confused (power, work, force, &c., being used synonymously as Boring and Titchener have pointed out). Further, Wrightson's final conclusions appear to be in actual error, for he claims that whereas when air is transmitting sound waves the air particles come to rest twice during each wave (at each crest and at each trough), the fluid of the cochlea, on the other hand, comes to rest four times, not only at each crest and each trough, but also each time the air particles pass through the mid position of their movement, i.e., each time the sine wave, representing the motion, crosses the axis. This conclusion, as Boring and Titchener point out, is at variance with the third law of motion, that action and reaction are equal and opposite. In view of this error of Wrightson, the remarks of Keith in reference to this matter become almost pathetic: "I had," Keith writes, "foolishly regarded the crests and troughs of sound tracings . . . as the points at which stimuli were produced, for at those points the ciliary movements appeared to be reversed. But I could not see how stimuli could be produced at the crossing points, until it was made clear to me by Sir Thomas Wrightson that the crossing points in the tracing of a sound wave are points at which there are equally great changes in pressure and movement." Yet Keith's original view was the correct one, and the view that Wrightson substituted was the incorrect.

Section 4 contains Wrightson's hypothesis of audition, which may be stated as two separate propositions as follows:—

(*a*) That when sound waves enter the ear, the whole of the basilar membrane is set into motion, and the different parts of the membrane move synchronously, rising and falling together by the same amount (p. 104). These movements of the basilar membrane cause to-and-fro movements of the hair-cells, relative to the membrane tectoria above them, and these at each change in direction of the movement cause impulses to pass up the auditory nerve.

(*b*) That the impulses passing up the auditory nerve occur four times during each complete air vibration, i.e., at each crest, trough and crossing point: and further that the time intervals between these impulse points are in certain cases approximately the same as those which occur in the individual tones which together are causing the air to vibrate. It is the recognition by the brain of these time intervals which causes the appreciation of the individual tones in a musical chord, the appreciation of overtones, harmonies and summation and different tones being similarly recognized.

About this second half of his theory (*b*) it may be said that it is this part which individualizes Wrights on hypothesis from other displacement hypotheses which have preceded it, e.g., from Rutherford's telephone hypothesis; but it is also this part which must be abandoned, since, as I have shown above, its supposed basis does not actually exist. The remaining features of the hypotheses are so like those of Rutherford's that the two must stand or fall together. Both hypotheses hold that the impulses travelling up the auditory nerve correspond in a definite manner in their time relationships and intensities to the air waves which set them up; both believe that the basilar membrane lacks any power of analysis; both relegate the ultimate analysis to the brain. The criticisms advanced against Rutherford's theory in the past therefore apply with equal force to Wrightson's, namely, that the initiation of nervous impulses at a rate which may reach 40,000 per second and their conduction up the whole length of the auditory nerve and through two different sets of synapses, and their final enumeration by the brain, are all processes which tax the credulity of the physiologist to the utmost. The classic experiments by which Rutherford sought to give his hypothesis something more than an imaginary basis are too well known to require description here: the grafting on to that basis of an additional hypothesis, as Wrightson has done, without at the same time any experimental extension, would seem to be a mistake.

Section 5 consists of Keith's appendix on the histology and morpho-anatomy of the organ of hearing, and comprises nearly half the book. For this essay I have nothing but admiration; it is absorbingly interesting, well written and full of that subtle knowledge that we should expect from the pen of a great anatomist. Whereas Wrightson disappointingly fails us at just those points where we should have expected an engineer to have made the surest guide, Keith serves us admirably not only when dealing with his own subject, but also when he digresses to physics and mathematics. If one has any single feeling which one would wish was not there, it is the regret that Keith had heard of Wrightson's hypotheses. Excellent as Keith's essay is, might it not have been better still with possibly not only Wrightson's hypothesis but also all others left out, a simple description of the parts of the inner ear without any idea in the background of hypotheses? Having considered above the different sections in turn, it remains to appraise the book as a whole. It is a book which in my opinion all interested in hearing should read; it contains one of the best descriptions of the comparative anatomy of the cochlea that I know, it contains many facts about the organ of Corti

which are not to be found elsewhere. We have Keith personally to thank for many of them. To Wrightson I would wish to say that although it has been my lot to criticize his hypothesis, yet I would like to express my admiration for the facts that he has brought to light about the organ of hearing and the clever way in which he has described them in his book.

H. HARTRIDGE.

The Psychology of Sound. By HENRY J. WATT, M.A., Ph.D., D.Phil., Lecturer on Psychology in the University of Glasgow. Pp. vii + 241. Cambridge: University Press, 1917.

This book is not very well named. It would be better described as: A theory about the nature of psychology illustrated by speculation concerning the character of auditory experience. Watt's first conviction is that psychology must occupy a position wholly independent of physics or physiology. His second is that all forms whatever of sensory experience must conform to a single scheme, and must illustrate the same structural principles. His third is that occupying an exceedingly important position among the attributes of sensory experience are what he calls "order," and something else of the nature of what other writers have called "extensity." It is on these assumptions, which are perfectly general, and are obtained by theorizing, rather than by experiment, that his account of audition is based. So, in the last chapter, we find him saying with the greatest honesty: "The theory expounded in this book has been devised by the writer not in the interest of a study of hearing as such, but for the sake of the more inclusive study of the whole range of sensory experience. This work on hearing is thus only an episode in a larger effort. It is my firm belief that all our sensory experiences can be completely accounted for in general systematic terms without our having recourse to the discoveries of physics or physiology at all." And what is written in words here is written in the spirit throughout the whole book.

An adequate review of Watt's volume ought therefore to begin by a thorough examination of his underlying assumptions. This cannot here be attempted, but one thing must be said. Whether in this realm or in any other, Watt cannot expect to gain many adherents to his views unless he will express his contentions in language more clear to the ordinary understanding.

Take the conception of "order" as an attribute of sensation, and thus of sound. It is introduced at the beginning of the book (p. 8). It occurs frequently all the way through to the very end (p. 215). Together with that of "volume," which is the name for the "extensive implication" of sounds, it bears all the burden of the theories put forward concerning tone, noise, fusion, consonance, the localization present in binaural listening, and many other matters. Yet, in spite of this, it remains throughout an elusive conception. The "ordinal differences of the elements of hearing," for example, "are the basis of the binaural localization of hearing" (p. 177), and "there is no

primary difference either physiologically or psychically between the binaural basis of localization and the uniaural basis of pitch" (p. 192). But pitch, which is thus a "kind of order," certainly is not to be supposed to be spatial (see pp. 28-29, 213, &c.), whereas localization, which is also "a kind of order," as certainly is. "Spatiality" is said to have "emerged" from "something in the single ear," this "something" being the ordinal, non-spatial attribute of the sound sensation. How it "emerged" is not clear, although the process is described as one of integration "with those ordinal differences of other senses that are dependent upon the physical positions of their stimuli."

In any case what "emerges" from the argument would appear to be that there are at least two kinds of order, one spatial and one non-spatial. Perhaps there may be any number of other kinds also. Perhaps, in point of fact, "order" as an attribute of all sensory experience simply means that within any such experience differences of some sort occur, and may form a basis upon which all kinds of subsequent discrimination proceed. Thus on p. 213 "order" is definitely spoken of as a "purely psychical arrangement." It would appear that material of *any* kind whatever might be so arranged. Positions, or moments, or qualities might all be "ordinal"; but it could not in the least be inferred from this that they were alike in any other respect than that they were an "arrangement." If this were literally all that was meant, although both pitch and localization could be spoken of as a "kind of order," it would by no means follow that the second "emerges" from the first. Nothing would follow, in fact, except that they would both be a kind of arrangement. This appears clearly to be inadequate. And it helps to show that in so far as the concept of "order" is to be of value in the solution of any particular problem ordinal differences must be treated as having a specific nature.

It is difficult to be certain about any of Watt's meanings. But it seems highly probable that "order," as belonging to sound, really signifies a good deal more than a mere arrangement. As it is actually made use of, the conception appears to be derived from a belief that tones are "volumic masses." That is, they are spread-out arrangements in which the various "atoms" have relative positions, and in which the "central atom predominates, thus giving the whole a pitch" (p. 206). Tone is a balanced mass of such atoms, noise an unbalanced mass. All problems of fusion are simply a question of the coincidence of volumes, and their resultant balance; while interval is mainly a question of the proportion between different parts of a "volumic outline or stress." Add "motion" to order and volume, and to Watt the structure of melody becomes clear. Study of the formation of scales brings us back to the same attributes combined and modified in various ways. As others have remarked, Watt's whole theory rests on his conception of volume, and this cenception is wholly speculative, having no empirical basis that can be discovered. Supposing now that it is right to treat order as involved somehow in volume, then, as the latter has avowedly extensive implications, it seems likely that, however much Watt may deny the fact, in some way or other a spatial character does lurk in order as the latter is here

attributed to sound. This would help to explain why Watt is certain that pitch differences are not qualitative, and might help to render the treatment of localization a little more intelligible. But whether in truth such a spatial character does belong to auditory sensations would still be a matter for dispute.

There is in the book a long chapter on "Physiological Theories of Hearing," in which summaries of all the leading theories are presented. These are of very considerable interest, though they are not always sufficient. Watt himself rejects the resonance hypothesis, as would be expected. He supposes that the basilar membrane is exceedingly elastic, and that to hear a sound means that an actual wave form is set moving along the membrane. A "physical tone produces a wave of depression of the basilar membrane beginning from the basis and extending along in proportion to its pitch, with a point of maximal depression in the centre round which relative intensities are arranged symmetrically and decreasingly." There is no evidence that this actually does happen. But that does not matter. For, experience being what it is—or what Watt alleges it to be—the assumption of the supposed physiological mechanism is perfectly consistent. If any investigation should be able to show that the structures involved could not react in the way proposed, then the investigation must be wrong. This is where the "independence" of psychology comes in.

The book shows upon every page evidence of the most careful research. But it is research by reading and thinking, not by experiment. Now every experimentalist in psychology knows that much of what chiefly convinces him is hard to put down on paper. And any man who relies mainly upon a critical reading of experimental work may fail to be convinced because some only of the data are set before him. In any specific psychological problem it is just as important for theorists to conduct experiments as it is for experimentalists to construct theories. In spite of his enthusiasm for his task, of the thorough way in which he has covered the field, and of the air of complete certainty with which he publishes his most interesting views, Watt must be said to have written a somewhat one-sided treatise. It is, in fact, philosophical rather than psychological. Perhaps it is consistent with all the facts. But a theory which no facts can confute may be of as little worth as one with which no fact can be made to agree.

F. C. BARTLETT.

The Form and Functions of the Central Nervous System. An Introduction to the Study of Nervous Diseases. By FREDERICK TILNEY, M.D., Ph.D., Professor of Neurology, Columbia University, and HENRY ALSOP RILEY, A.M., M.D., Associate in Neurology, Columbia University. Pp. 1,020, with Glossary and Index. New York: Paul B. Hoeber, 1921.

Within recent years no branch of medical science has made more rapid progress than neurology. The work of investigators on the normal and

abnormal histology and physiology of the nervous system in men and animals has led to the accumulation of a vast amount of knowledge, which is scattered widely throughout the literature of different countries and is not readily accessible. There is therefore increasing need for the co-relation and presentation in book form of this wealth of data and of the main speculations which have arisen from them upon the structure and functions of the nervous system. It is this task that the authors have attempted; but unfortunately the result of their labours has not been followed by the success that might have been hoped for.

They say in the preface that their book is intended for the use of students, as an introduction to the study of nervous diseases; but, as it contains upwards of a thousand pages of text, it is obviously much too large for this purpose. What is required by the student in his medical course is a clear and concise account of nervous structure and function presented in a form in which it can readily be applied to the study of disorders of the nervous system. It should not be encumbered with debatable points of detail or an ambiguous and misleading terminology. As far as possible it should give the facts and show how they can be united by accepted general principles.

If, on the other hand, the book is to be regarded as a comprehensive treatise for reference, as its size might indicate, it falls short in many ways. For example the bibliography is never referred to in the text, a want which is all the more felt as the authors tend to dogmatize on controversial questions. The perpetual insistence on syndromes is trying to the reader, who is anxious to discover the significance of some sign or symptom. For instance "the Babinski reflex" is repeatedly mentioned, but there is no coherent or systematic account of current views as to its nature and origin. The term "sensory" is employed to designate both sensory and non-sensory afferent fibres and impulses, a usage which inevitably gives rise to considerable confusion. The information given is extensive, but is insufficient on several important points both of structure and function. Thus, the fasciculus retroflexus of Meynert is scarcely mentioned and vision and speech are inadequately dealt with. To illustrate the disorders of function, which result from lesions of different parts of the nervous system, the authors have drawn freely from their clinical records; but the value of these examples is often diminished by the incompleteness of the observations and, occasionally, by the diffuseness of the morbid changes.

The meaning is not infrequently obscured by the use of barbarous terms such as "arthresthesia," "piezesthesia," &c., which are unnecessary and have not received general acceptance. The ear of the reader is jarred by "juxtagriseal," "behavioural" and "tractability" and the proofs have been carelessly read. "Deiters" and "Gowers" are correctly spelt in the index, but are habitually wrong in the text; the name of the French neurologist becomes "Déjérine" in the index, "Djerine" on three of the plates, but is rightly printed in the references.

Of the 591 figures, containing 763 illustrations, many are good, but others,

especially the reproductions of photographs of sections of the nervous system, could be considerably improved.

We have been somewhat lavish in criticism because we believe that an authoritative and comprehensive presentation of the available knowledge on the applied and comparative anatomy and physiology of the central nervous system is much needed. This book will probably pass into a second edition, and if so there is no reason why it should not be greatly improved to meet the want.

Manic-depressive Insanity and Paranoia. By Professor EMIL KRAEPELIN, of Munich, translated by R. MARY BARCLAY, M.A., M.B., from the eighth German edition of the "Text-book of Psychiatry." Vols. III and IV. Edited by GEORGE M. ROBERTSON, M.D., F.R.C.S.Edin., Professor of Psychiatry in the University of Edinburgh and Physician to the Royal Asylum, Morningside. Pp. 280. Edinburgh: Livingstone, 1921.

To Professor Kraepelin is due the credit of recognizing manic-depressive insanity as a single morbid group characterized by profound alterations in emotional tone, but unaccompanied by progressive mental deterioration. The phases of exaltation or depression may be of very different intensity; thus a patient, who suffers from severe recurrent attacks of melancholia, may show symptoms of hypomania so slight that they are considered as the natural rebound of convalescence. Conversely, a man who becomes excited and troublesome, and on recovery passes into a quiet unhappy state, may appear to be brooding over the consequences of his outburst of folly. But the most difficult cases from the point of view of the neurologist are those usually called "recurrent neurasthenia"; each attack is attributed to some worry or physical strain, and the essential nature of the condition and its close relation to manic-depressive states is not recognized. Careful inquiries will, however, elicit the suddenness of the onset and its want of direct dependence on emotional shock. This morbid condition persists for many months or even years and then passes away as suddenly and apparently as causelessly as it came.

We therefore welcome this translation of the chapters in Professor Kraepelin's book dealing with manic-depressive states in all their forms, straightforward circular insanity, recurrent excitement or depression, repeated attacks of "neurasthenia," or mixed psychical conditions in which all these forms coexist to a greater or less degree. No cases are described, but each chapter is illustrated by a multitude of instances, which tend somewhat to obscure the general clinical picture. Every aspect of this protean malady is considered systematically: but at the end Professor Kraepelin confesses that he is completely ignorant of its true nature and cause.

The last two hundred pages are devoted to an account of Paranoia, in the narrower meaning of the term, as a primary form of insanity, which develops insidiously and leads to no permanent psychical weakness. Here, in these

chapters more clearly than elsewhere, we see the difficulties produced by a classification built up solely on overt symptoms and deviations of conduct.

The translation is accurate but clumsy: the book is full of German phraseology and it is a pity that some of the legends on the figures have been left in the language of the original. English students who require a translation of so well-known a work may be puzzled by such explanations as "Gesunde richtig, Gesunde falsh," "Zornige Manie," "Depression mit Reizbarkeit," and the numerous long German citations.

Anxiety Hysteria. Modern Views on some Neuroses. By C. H. L. RIXON, M.D., M.R.C.S., and D. MATTHEW, M.C., M.B., Ch.B. Pp. 124. London: H. K. Lewis, 1920.

The authors have attempted to express in simple language their views upon the psychopathology, symptomatology, diagnosis and treatment of the more common forms of the psychoneuroses of war. They address especially the general practitioner who, as a medical referee or a member of a medical board under the Ministry of Pensions, has frequently to deal with such patients; and as a practical guide to investigation and to rational methods of treatment on broad lines it will serve a useful purpose.

A chapter is devoted to psychopathology and some of the mental processes concerned in the production of symptoms are explained. Diagrams are freely used and will perhaps be found helpful, although they are apt to lead to somewhat stereotyped notions of the processes they are intended to elucidate. The views expressed agree with those of most observers, that the conflicting forces at the base of war neuroses are those connected with the instinct for self-preservation and with the sentiments linked round the ego-ideal. The terminology employed is as a rule clearly defined but it would have been advantageous to have used separate terms for voluntary and involuntary forgetting.

Psychology and Psychotherapy. By WILLIAM BROWN, M.D., D.Sc. Pp. 196. London: Edward Arnold, 1921.

This little book deals in an easy and popular manner with the various aspects of modern psychopathology. The introductory chapters are devoted to dissociation and to the theories evolved to explain hysteria and the functional neuroses. The second part contains a clear statement of the teaching of Freud, with a short description of psycho-analysis and word-association. This is followed by a chapter on psychotherapy and a section on the lessons of war. The style is clear and simple and the book can be recommended to those who are anxious to know something of the diverse views now current on the processes responsible for the psycho-neuroses.

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Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I to XXIII inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

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EDITOR.

[JULY, 1921.]

BRAIN.

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LESIONS OF THE BRACHIAL PLEXUS ASSOCIATED WITH RUDIMENTARY RIBS.

BY PERCY SARGENT, C.M.G., D.S.O., F.R.C.S.

INTRODUCTORY.

RUDIMENTARY ribs at the upper thoracic operculum have for centuries attracted the attention of anatomists. According to Keen [12], whose paper should be referred to for the historical aspect of the subject, the earliest account of such an anomaly is that given by Hunauld in 1740, and many descriptions of sporadic specimens are scattered throughout the old anatomical and surgical literature. The advent of evolutionary ideas drew to them the attention of morphologists, some of whom attempted to show that the "general shortening of the thoracic region, which is at work throughout the mammalian series, was evidenced as a distinct and ordered evolutionary process from ape to man" (Wood Jones) [9]. The studies of Dwight [5] and others failed to confirm this view, and Dwight concluded that these costal and vertebral anomalies are "merely variations round a mean, which for want of a better word we must call accidental."

Were it not for the correlated abnormalities of the soft parts, and the symptoms which may arise by reason of this association, these rudimentary osseous structures might have failed to attain a position of any practical interest. When aneurysms were common and occupied much of the surgeon's attention, an accessory rib was regarded as a possible cause of that condition, or as a convenient *point d'appui* for pressure in its treatment. It was also recognized that such a rib might give rise to difficulty in diagnosis. Thus Paget [17] wrote: "The imitation of subclavian aneurysm is made more marked when a nervously pulsating subclavian artery lies upon a cervical rib." It is only comparatively recently that the importance of these anomalies from the neurological point of view has been fully recognized.

Patients suffering from symptoms due to a rudimentary rib constitute a clinical group of not inconsiderable size, to which we are in a position to transfer a number of cases from amongst those bearing such "diagnostic" labels as brachial neuritis, brachial neuralgia, uniradicular palsy, progressive muscular atrophy, writer's cramp, occupation neurosis and symmetrical atrophy of the hands. Thus Farquhar Buzzard [3] in 1902 described six cases of nerve-root lesion under the title of "Uniradicular Palsies of the Brachial Plexus." A review of the clear and accurate account of these cases cannot but convince one that four were instances of the lesion which would now be ascribed to "cervical rib." Similarly, as pointed out by Bramwell and Dykes [2], Lewis Jones [11] reviewed in 1908 a group of cases to which he had drawn attention in 1893 under the title of "Symmetrical Atrophy affecting the hands in young people," and found a well-marked cervical rib to exist in no fewer than ten out of fourteen patients of that group.

Relation between Costal Anomalies and the Nerves constituting the Brachial Plexus.

Wood Jones [9] has demonstrated the fact that costal anomalies are associated, as he believes causally, with variations in the constitution of the brachial plexus. He believes that the development of ribs both in the cervical and lumbar regions is arrested at the points where the nerves, taking an oblique course in order to reach the limbs, cross the primitive mesoblastic costal elements, thus giving rise to the "ribless neck and waist." The first indication of this interrelation is to be found in the neural grooves upon the first rib. The first thoracic root[1] (or the lowest cord of the brachial plexus resulting from its junction with the eighth cervical) in passing to the axilla causes, by pressure, the groove upon the first rib which, from being supposed to lodge the subclavian artery, is commonly known as the sulcus subclaviæ; this groove he consequently prefers to call the sulcus nervi brachialis. When the first thoracic root is very large, the groove may be so strongly marked that the rib is actually bent downwards at this point. When a large part of the second thoracic nerve goes to the brachial plexus, the first rib may actually be deficient, and may be either continued on to the sternum as a fibrous cord, or pressed down upon and fused with the second rib so as to constitute a "bicipital rib."

[1] Where the term "root" occurs, it indicates that part of the anterior primary division of the spinal nerve which enters the brachial plexus.

A brachial plexus so constituted is a post-fixed plexus. The conclusion is therefore that the more post-fixed the plexus, the more likely is the first rib to be ill developed or abbreviated. Conversely, a pre-fixed plexus is likely to be associated with an abnormal development of the costal element of the seventh cervical vertebra, and there are many facts which lend support to this view. It is true however only within certain limits. Wingate Todd [22], as the result of his observations, concludes that "cervical ribs may be present in cases where the composition of the brachial plexus is normal" and that "the disposition of the nerve trunks alone is insufficient in many cases to account for the presence and length of rudimentary ribs." During many of my operations I have attempted to make a careful examination of the plexus so as to determine its composition. On one occasion when I could find no contribution from the first thoracic nerve, I stimulated the eighth cervical root and caused contraction of all the intrinsic muscles of the hand. In other instances, however, the contribution from the first thoracic nerve has been evident, and apparently of normal size. Similar discrepancies may be noted in the descriptions of dissections which are to be found scattered throughout anatomical literature. In this connection the description given by M. F. Lucas [14] of the composition of the brachial plexus in two dissecting-room subjects is particularly interesting. In a female subject the left rib, 6 cm. long, was attached by a short fibrous band to the medial border of the first rib, and was grooved by the lowest and middle trunks of the plexus. The fourth cervical nerve contributed to this plexus, which was only slightly prefixed. On the right side the cervical rib was only half as long, and here the fourth cervical nerve made no contribution to the plexus, whilst the contribution of the first thoracic nerve was appreciably larger than on the other side. In a male subject, each cervical rib was a little over 4 cm. long, and in both cases the composition of the brachial plexus was normal. Hertslet and Keith [7] give a drawing showing side by side, for comparison, one thorax with a rudimentary first thoracic rib and another with a cervical rib. The first is associated with a postfixed plexus and the second with a prefixed plexus. In the case of the rudimentary thoracic rib a large part of the second thoracic nerve joins the whole of the first thoracic, the combined nerve passing over the bony part of the rib. In the case of the cervical rib, a small branch from the first thoracic nerve passes up to join the eighth cervical, the combined nerve passing over the bony part of the rib. This plexus receives a large branch from the fourth cervical

nerve. Arbuthnot Lane recorded a case of rudimentary first thoracic rib with a definitely postfixed plexus. Describing a specimen of cervical rib he notes that the "firm tendinous cord" which continues the line of the cervical rib is attached to the upper margin of the first thoracic rib and its cartilage.

It seems quite clear therefore that although, generally speaking, some prefixation of the plexus is usually to be found associated with cervical ribs, and some degree of postfixation with abnormal first thoracic ribs, yet the relationship is by no means constant. Nor does the form and size of the abnormal rib appear to bear any constant relation to the composition of the plexus.

Were the plexus always prefixed for a complete segment when a well developed cervical rib is present, we should expect, other things being equal, that no symptoms would occur. On the other hand, a large contribution from the first thoracic nerve, by passing over the accessory rib or its fibrous continuation, would be more exposed to injury, and symptoms would be more likely to arise. Prefixation of the plexus would thus be an advantage in the presence of a cervical rib.

Classification of Cervical Ribs.

The classification of Gruber, quoted by Keen, is as follows:—

"*First degree*, a very slight increase of the costal process, not reaching beyond the true transverse process. *Second*, a rib protruding beyond the transverse process to a moderate extent and ending either free in the tissues or attached in some way to the first thoracic rib. *Third*, a still further degree of development in which the cervical rib extends for a considerable distance toward or even to the cartilage of the first thoracic rib, possesses a complete body, and is united directly or by means of a ligament with the cartilage of the first rib. *Fourth*, a complete cervical rib uniting at the anterior extremity with the cartilage of the first rib through which it reaches the manubrium."

This however does not embrace all the forms which may be met with, and takes no account of whether or not the rib is jointed to, and therefore to some extent movable upon, the seventh cervical vertebra. It therefore requires amplification.

The actual length and shape of the bony portion of the rib does not seem to have any great clinical significance; its direction, on the other hand, is important, as was first pointed out by Thorburn [21]. This fact is well illustrated by a remarkable case of my own (Case 31).

where the rib, after subperiosteal resection, was reproduced, but, being moulded during its reincarnation in a direction less at variance with that of the nerves, the symptoms previously present did not reappear. A somewhat similar case by Weber is quoted by Hinds Howell [8]. Subperiosteal resection of a cervical rib was followed by regression of the symptoms which, however, reappeared with the re-formation of the bone. Subsequent removal resulted in recovery.

The mobility of the rudimentary rib may also influence the symptoms, so that the jointed rib, whatever its length, deserves separate notice.

The types which I recognize are as follows :—

(1) An exaggerated costal process of the seventh cervical vertebra, not jointed to but fused with the transverse process, and continued forwards and downwards as a fibrous band to be attached to the first thoracic rib behind the scalene tubercle.

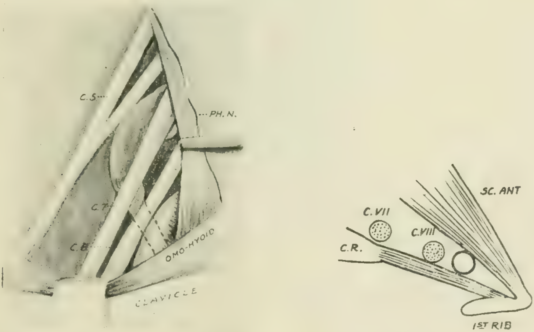


FIG. 1.

Fig. 1 represents an example of Type 1, where the bony rib is unusually long for a non-articulated rib. The diagram alongside indicates in section the relation of the nerves and artery to the abnormal costal elements.

(2) A short rib, articulated to the seventh cervical vertebra by costo-central and costo-transverse joints, and continued onwards as a fibrous band to be attached to the first rib as in type 1.

(3) A jointed rib of sufficient length to carry the eighth root upon its bony portion, and attached by a fibrous band to the first rib.

(4) A jointed rib of which the anterior extremity makes contact with the first thoracic rib, the two being either fused, or united by an irregular articulation.

(5) A rudimentary first thoracic rib, the anterior fibrous portion of which is attached to the sternum, usually by a rudimentary costal cartilage.

Varieties doubtless exist amongst these types. This is exemplified by the following instance which I am unable to place in the foregoing classification (fig. 18). *Case* 28: The rib was articulated both with the body and transverse process of the seventh cervical vertebra. The seventh and eighth cervical roots lay upon the bony rib, being separated by a well-marked ridge. The anterior extremity was continued on towards the anterior end of the first thoracic rib, passing in front both of the subclavian artery and the first thoracic nerve. Hinds Howell [8] mentions a similar case operated upon by Rawling in which the rib projected between the eighth cervical and first thoracic roots which united beyond it.

Relation of the Spinal Nerves to Cervical Ribs.

The relation of the roots of the brachial plexus to the various types of rudimentary ribs appears to be fairly constant, and is indicated in the diagrams. The observations made during the course of an operation cannot however be as accurate as those made in the dissecting room, and the point least easily ascertained by operative dissection is the size of that portion of the first thoracic nerve which enters the plexus, and its exact relationship to the abnormal costal element. One cannot always determine therefore whether the lowest nerve seen is the eighth cervical alone, or the combined trunk of the eighth cervical and first thoracic roots. It may however be stated quite confidently that the commonest arrangement is for the seventh root to lie upon the bony part of the cervical rib, and for the eighth root (or the lowest cord of the plexus) to be carried upon the fibrous portion. Further, my observations have convinced me that, whilst the contribution of the first thoracic nerve to the plexus may be of considerable size, it is usually small and may even be absent. This confirms the view that when a cervical rib exists the plexus tends to be of the prefixed type. It is certainly not true however to say that the better developed the cervical rib the more prefixed is the plexus, or vice versa. A rudimentary first thoracic rib is a much rarer anomaly than a cervical rib, but it seems to be an established fact that in such cases the plexus tends to be of the postfixed type. This is in harmony with the observation of Wilfred Harris [6] that the postfixed is not so common as the prefixed type in man.

The Sympathetic Nerves.

The main sympathetic nerve supply to the upper limb comes from the inferior cervical and first thoracic ganglia, usually by two trunks which enter the first thoracic and eighth cervical nerves close to their foramina of exit from the vertebral column. A communication, stated by Cunningham to occur in 70 per cent. of cases, from the second thoracic nerve joins the first thoracic, and contains sympathetic fibres. Wingate Todd [22] has given a diagram of this arrangement, and has pointed out how the sympathetic nerves to the upper limb may be subjected to injury in the presence of a cervical rib.

Dissections made by Professor F. G. Parsons and myself show the sympathetic branches entering the brachial nerves (fig. 2). I have

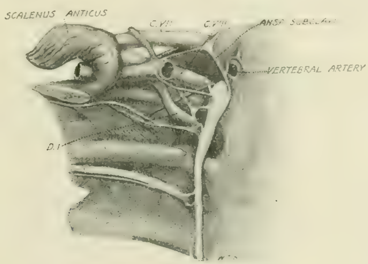


Fig. 2.—From a dissection by F. G. Parsons, showing sympathetic branches to the brachial plexus.

attempted to trace these nerves after their incorporation with the main trunks. Dr. W. Penfield has made serial sections which show that bundles both of small medullated and of non-medullated fibres tend to lie, at first at any rate, in the periphery of the mixed nerve. It may be that this arrangement is very variable, and that in those patients who exhibit well-marked vasomotor symptoms the sympathetic fibres are specially exposed to injury from their superficial position. As regards oculo-pupillary symptoms, the only instance which I have encountered where such were present occurred in a woman 42 years of age. It was noted that the right pupil (the affected side) was smaller than the left, and that the right palpebral fissure was sometimes wider than the left. When seen six years after operation no such symptoms could be detected, and the arm had completely recovered.

Thorburn [21] states that he never saw oculo-pupillary signs, and that the only case in Schönbeck's series had co-existent syringomyelia.

The Subclavian Vessels.

The height to which the subclavian artery rises in the neck varies considerably in normal individuals, and is naturally affected by the position of the shoulder. Many patients with symptoms due to cervical rib are "bottle necked," and the droop of the shoulders tends to exaggerate the height to which the artery rises above the clavicle. In some recorded instances the artery has been found passing over the accessory rib. I have not encountered an example of this condition amongst the patients upon whom I have operated. The only case in which I have seen the artery passing over an abnormal rib was an example of rudimentary first thoracic rib. In almost all my cases the artery has been found either lying upon the first thoracic rib, or just held off from it by the fibrous band. In two instances a very large transversalis colli or suprascapular artery has been found lying upon and grooving the cervical rib. The pulsations of this vessel had been noted before operation, and might easily have been mistaken for those of the subclavian artery.

The varying relation of the artery to abnormal ribs described by Wingate Todd [22] corresponds closely with my own observations.

In a recent case of my own, a well marked example of Type 4, the course of the subclavian artery was so peculiar as to deserve special mention (fig. 3). A large accessory rib, which formed a remarkable prominence in the supraclavicular fossa, was joined by an irregular articulation with a mass of bone upon the upper surface of the first rib. The subclavian artery rose high in the neck to the inner side of the accessory rib, and then, bending downwards, grooved the inner aspect of the mass of bone formed partly from the cervical rib and partly from the first thoracic rib. It then turned outwards just below the bony mass, to reach the axilla. There was no contribution to the plexus from the first thoracic nerve. The lowest cord and the subclavian artery were thus separated by a mass of bone, and over this there had developed a well-marked synovial bursa.

A number of cases have been reported as having had aneurysmal enlargements upon the artery. The subject is fully discussed by Keen [12] and need not be further considered here. At most of my operations the artery has been carefully observed, and in only two was anything resembling a dilatation seen. Both were examples of Type 4,

the cervical rib being attached to the first thoracic rib by a mass of bone against which the artery lay. In one the artery was dilated proximal to the mass, and in the other a fusiform dilatation existed at the point of contact. In the latter no radial pulse could be felt; in the former the pulse disappeared with the arm in the dependent position. In a third case, belonging to the same type, the artery was definitely narrowed at the point where it crossed the bony mass resulting from the fusion of the cervical with the thoracic rib. In this patient no radial pulse could be felt in any position of the limb; a systolic bruit was to be heard, and a thrill could be felt, above the clavicle. The absence of the radial pulse in the dependent arm is but an exaggeration of a common phenomenon in cases of cervical rib.

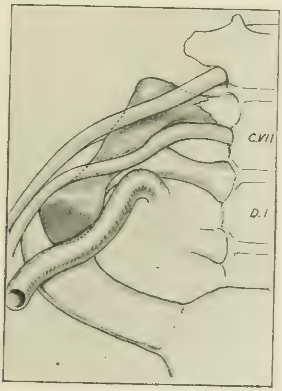


FIG. 3 is an example of Type 4, with a definitely prefixed plexus, and the subclavian artery in peculiar relationship.

When the arm is hanging by the side, the least downward traction will often cause the radial pulse to disappear. In many instances of bilateral cervical rib I have noted that the radial pulse can more easily be made to cease by this means on the side of the most pronounced nerve symptoms. Doubtless the artery is, in these cases, subjected to the same mechanical strains as is the lowest cord of the brachial plexus. Similarly, a systolic murmur, not conducted down the arm, can sometimes be heard just above the clavicle, and can be made to vary with the position of the arm. The subject of arterial thrombosis and gangrene of the fingers is also fully discussed in Keen's

paper, to which reference should be made. No such case has come under my own observation. "Vascular symptoms" have, in my patients, always been due to vasomotor disturbances.

The subclavian vein is rarely obstructed, and indeed its position is such that it can scarcely come into relation with any part of any cervical rib. Thorburn mentions one case in which he believed that the œdema might have been due to pressure on the subclavian vein.

Causation of the Symptoms.

The factors which determine the onset of symptoms in persons with cervical ribs are so excellently presented in a recent paper by Bramwell and Dykes [2] that a full discussion is unnecessary here.

That the lesion is traumatic cannot be doubted. In the cases of sudden onset of symptoms some definite strain has occurred, such as lifting a heavy weight, which would naturally bring nerve and "rib" into sudden and violent contact.

When the symptoms are of gradual onset the damage is, I believe, produced by the continual friction of the nerves against the non-ossified costal process, caused by the movements of the arm and by the respiratory movements. I have frequently been able to demonstrate, during an operation, the manner in which the nerve is pushed upwards and rolled over as the band tightens during inspiration and in certain positions of the arm. Further, it can easily be seen that as soon as the band is excised the nerve at once sinks to a lower level in the wound and is relieved of all tension. I believe the mechanism to be commonly as follows: The fibrous band, springing from the costal process of the seventh cervical vertebra, is usually attached to the upper surface of the first thoracic rib behind the axis of movement of that rib. As the anterior end of the rib rises in inspiration, the posterior part tends to sink, so that the band is made taut, and comes into harmful contact with the nerve which lies against it. Thomas and Cushing [19], in 1903, suggested that the fibrous band was the cause of the symptoms. Here "the lowest cord of the plexus was compressed by a dense fibrous band which passed from the tip of the rudimentary rib under the plexus to its point of attachment to the first thoracic rib. The band therefore over which the lowest cord of the plexus arched at rather an acute angle was the offending agent rather than the bony projection itself."

Whether any gross histological changes occur in the damaged nerve,

such as can be demonstrated in an ulnar nerve similarly affected by rolling movements over an abnormal humeral condyle, I have had no opportunity of ascertaining, but one would naturally suppose that such changes would occur, and that they would be the more pronounced the longer the symptoms had persisted. This supposition is supported by the fact that the rapidity and degree of recovery after operation are to a large extent related to the previous duration of the symptoms. On the other hand, I have never encountered any instance of palpable thickening, nor have I seen any naked-eye evidence of damage, except in one case in which a distinct groove marked the nerve at the point where it had been in contact with the band.

I have now operated upon sixty-five patients for the relief of symptoms due to a "cervical rib." Whilst the ribs were always bilateral, they were not invariably symmetrical, and when any conspicuous difference in size existed the symptoms were most frequently worse on the side of the smaller rib. The right arm was wholly or chiefly affected in thirty-three patients and the left in twenty-four, whilst eight patients presented well marked bilateral symptoms.

The age and sex incidence in my cases conform with those noted by other observers. Of sixty-two patients the average age was thirty-five ; fifty-five were females and seven males.

With the exception of three school children (aged respectively 12, 13, and 15), one artist, and one patient who might be classed amongst the "idle rich," all were engaged in active and often strenuous pursuits. Amongst the males there were two painters, one medical practitioner, one soldier, and one stoker. Of the females there were thirty-two employed in domestic work, five clerks, five nurses, three seamstresses, two pianists, two shop assistants, two telegraphists, one typist, one letter sorter, one teacher, and one milk-carrier.

Symptomatology.

The symptoms which may be caused by a cervical rib are well known. Pain, paræsthesia, disorders of circulation, affections of cutaneous and other forms of sensation, weakness, wasting, and alteration of electrical reactions, occur in varying combination and in varying degree in individual cases. These symptoms may be grouped into those of damage to :—

(1) Somatic afferent fibres, namely, neuralgic pain, and disturbances of cutaneous and deep sensibility.

(2) Somatic efferent fibres, namely, weakness, wasting and alteration of electrical excitability in the affected muscles.

(3) Sympathetic fibres, namely, circulatory disturbances (coldness, cyanosis, œdema) and certain paræsthesiæ (tingling, numbness, and feelings of coldness or swelling).

Counting each case with bilateral symptoms as two, the order of frequency of the symptoms for which the patients seek relief is (in fifty-eight cases) as follows :—

(1)	Weakness	48 = 79	per cent.
(2)	Aching or paroxysmal pain	42 = 72	,,
(3)	Paræsthesia	24 = 39	,,

Muscular wasting was evident in forty-three (72 per cent.), objective sensory loss in twenty (34 per cent.), and signs of vasomotor disturbances in seven (12 per cent.).

The " weakness " of which so many patients complain is sometimes described as " clumsiness," or inability to perform the finer acts demanded of the hand, especially after the hand has been in use for some time. Thus writing, sewing, typewriting, and piano playing may be rendered difficult or impossible. This symptom is often complained of by those with no visible muscular wasting, and is presumably dependent upon a disturbance of deep sensibility. In early cases it is inconstant, only occurs as the result of exertion, and quickly passes off with rest. We find that sixteen out of fifty patients were engaged in pursuits requiring the constant use of the hands for fine and specialized movements. We may no doubt add to these a certain number of the patients engaged in domestic work, for much needlework falls to the lot of a good many of them.

The Affected Muscles.

Kinnier Wilson [24] was the first to draw attention to the fact that some patients with cervical rib exhibit a characteristic peculiarity in the muscular wasting. This peculiarity consists in the picking out of abductor brevis and opponens pollicis, the other thenar muscles being relatively or absolutely unaffected. He concludes that the thenar muscles derive their nerve supply from two roots, namely, the abductor and opponens from the seventh and the flexor brevis pollicis from the eighth cervical.

With a normal arrangement of the brachial plexus the hypothenar muscles derive their nerve supply from the first thoracic root, the interossei chiefly from the eighth cervical, and the superficial flexor

muscles of the forearm from the sixth cervical. In the thirty-two cases of my series belonging to Types 1 and 2 which exhibited muscular wasting, all the intrinsic muscles of the hand and the superficial flexors of the forearm were affected six times; all the intrinsics of the hand (without forearm involvement) fourteen times; whilst in twelve instances the muscles chiefly affected were the abductor and opponens pollicis. In terms of normal roots this would mean involvement of—

C6, 7, 8 and T1 in six,
C7, 8, and T1 in fourteen,
and C7 and 8 in twelve.

Thus C6 would be involved in six, and T1 would be left out in twelve, C7 and C8 being affected in all. We are, however, dealing with plexuses of varying composition, but with a tendency to prefixation, in which the contribution from first thoracic nerve is small or absent. The muscles of the hand are therefore in these cases chiefly supplied by the eighth and seventh roots, which are those most exposed to the traumatism of the commonest variety of cervical rib.

Theodore Thompson [20], commenting upon the difficulty of understanding why a seventh cervical rib should cause symptoms referable to the first thoracic root, called attention to the varying constitution of the lowest cord of the plexus associated with cervical ribs.

The Sensory Changes.

In all cases cutaneous sensibility to cotton wool and to pin-prick has been carefully tested. Unfortunately the other forms of sensibility have been too rarely investigated with accuracy to permit any general statement being made, but impairment both of thermal and of postural sensibility have been noted several times, in addition to those of pain and touch.

In 60 per cent. of my cases no alteration of sensibility either to cotton wool or to pin-prick was detected. Whether more accurate testing might have revealed some abnormality it is impossible to say, but at any rate it can be stated definitely that in at least half the cases which have come under my own observation any sensory changes which may have existed were too slight to be detected by the ordinary clinical means of examination.

In twenty cases carefully charted, there was dulling of sensibility to pin-prick and cotton wool, distributed regionally as follows:—

Ulnar border of arm, forearm and hand	.. ..	3
,, ,, forearm and hand	.. ..	4
,, ,, forearm ..	.. ..	3
,, ,, hand ..	.. ..	5
Radial border of arm and forearm	.. ..	1
,, ,, forearm and hand	.. ..	2
,, ,, hand ..	.. ..	2

According to the commonly accepted delimitation of root areas, we should have concluded that the roots most often affected were, in order of frequency, the first thoracic, the seventh cervical, the second thoracic, and the sixth cervical, whilst the eighth cervical nearly always escaped.

Let us however review the forty-four cases belonging to Types 1 and 2, where the seventh root rests upon the bony part of the rib and the eighth (or lowest cord) is carried upon the fibrous band. In only fourteen of these was any sensory change detected, and in twelve of them its distribution corresponded with the usual distribution of the first thoracic root—in two with that of the seventh cervical. We are not however dealing with a normal plexus, but with a plexus of the prefixed type in which the area usually supplied by the first thoracic root tends to be supplied by the eighth cervical, namely, that which is most exposed to the traumatism of the fibrous band. In the majority of cases therefore the distribution of the cutaneous alteration of sensibility comes into line with the anatomical arrangement observed at operation.

In this connection attention may be called to two particular cases. In one (Case 12), the area of diminished sensibility to pinprick and cotton wool embraced only the thumb and the terminal phalanges of the index and middle fingers, whilst the only muscle affected was abductor brevis pollicis. Here the eighth cervical root passed over the tight band, whilst the first thoracic, passing beneath the band, escaped injury. In the second case (Case 49) the area of diminished cutaneous sensibility embraced the radial half of the forearm, both behind and in front, together with the thumb, index, and middle fingers; there was no muscular wasting. In this case the first thoracic nerve made no contribution to the plexus. It cannot be said that these cases furnish, either from the distribution of the sensory changes, or the muscles affected, any reliable information of morphological value regarding the normal distribution of the roots of the brachial plexus. Not only is the degree of shifting of the plexus upwards or downwards upon the cord variable, but the position which the different bundles of fibres occupy in the individual spinal nerves is probably subject to much variation.

The bundles which are situated nearest the surface of the nerve at the point of contact with the abnormal rib would presumably suffer most severely.

A recognition of the usual relation of these variable plexuses with the various kinds of cervical rib is, however, important from a practical point of view. In 1903 Edwin Bramwell [1] reported a case the salient features of which were as follows: pain down the inner side of right forearm for about three years; gradual loss of power in the hand, with cold feeling, for about twelve months; wasting of thenar and hypothenar muscles together with pronounced weakness of the flexors of the fingers; an area of impaired sensibility on the ulnar side of the forearm from just above the elbow down to the wrist. Having made a diagnosis of a lesion of the first thoracic root before its junction with the eighth cervical, he writes: "It is interesting to note the intimate relation of the first dorsal nerve-root to the sharp internal border of the first rib. Such a relation can hardly fail to predispose this root in an especial manner to traumatism." The suggestion of a first rib, rather than a cervical rib, being the cause of the symptoms in a case of this character, thus made for the first time, has been acted upon, and in several cases Stiles has removed a portion of the first rib, with excellent results. Similar cases are recorded by Morley [15], Murphy [16] and others. Stopford and Telford [18] have fully recorded and discussed seven examples of a similar nature. In their first case "a very large contribution from the first dorsal root was found ascending in a deep groove on the inner border of the first rib to join the eighth cervical root." There was a small cervical rib on the other side, but none on the affected side. In another of their cases "the contribution from the first dorsal segment of the cord was unusually large." They express the opinion that "a predisposing cause seems to be the anatomical relation of the nerve trunk and the rib, when the bone is bevelled by a trunk which receives a large contribution from the upper two dorsal roots."

In the light of these reports I have reviewed my own cases of cervical rib. Amongst them are several cases presenting symptoms of lesion of the first thoracic root in which the radiographic evidence of cervical rib was negative. In one such instance the note made at the operation states that a strong fibrous band, over which the eighth root passed, tightened in a very conspicuous manner with each inspiration, and that on its detachment from the tip of the seventh cervical costal process it was immediately pulled downwards for fully half an inch.

This patient was cured and remains well after an interval of nine years. I believe that the same good result would have followed Stiles' operation of removal of a portion of the first thoracic rib, carrying with it the lower attachment of the non-ossified "cervical rib." I believe, too, that in some cases the more complete relief of tension which Stiles' operation affords would probably render it the procedure of choice, and I am inclined to think that some of my failures might for the same reason have been successes had the larger operation been done. Stiles' operation, however, is designed for relieving the first thoracic root from the pressure of a normal first thoracic rib. In such cases one would expect that the plexus would tend to be to some extent postfixed, and the cases would fall into a class approximating to that which is characterized by a rudimentary first thoracic rib with a frankly postfixed plexus.

TREATMENT.

All the symptoms which may be caused by a cervical rib are capable of relief or cure. It must not be forgotten that these are common anomalies, and just as cervical ribs frequently exist without causing any symptoms whatever, so also they are likely to be found in patients presenting similar symptoms due to some totally different cause. Such indeed is the case, and unless the greatest care is exercised there is considerable risk that futile operations may be performed on patients with syringomyelia, progressive muscular atrophy, toxic neuritis, and other conditions wholly unconnected with the co-existent cervical rib. On the other hand there is perhaps even more risk that patients whose symptoms are due to such a costal anomaly may be denied the prospect of relief on account of failure to recognize the fact that "neuritis," "brachial neuralgia," and certain occupational disabilities are so often directly dependent upon rib-pressure. In cases of real doubt it is certainly legitimate to afford the chance of relief offered by an operation which, gently and carefully performed, is almost entirely free from risk.

Operation is not called for in all cases. In some a change of occupation is sufficient; in others a greater use of the other arm to relieve the affected side. The wearing of a sling is often beneficial. Yet again, the development of the muscles which support the shoulders by massage, muscle-training, and other means is a useful line of treatment. Patients however who are obliged to continue following certain occupations, and who cannot give up the time necessary for muscle

training, should be submitted to operation. Other forms of treatment, such as those mentioned, may be required in addition.

The details of the surgical operation need not be given here. Suffice it to say that its object is not merely to remove an abnormal bone, but to divide or excise any non-bony fibrous structure against which the nerves are being damaged. In one case, for example, a clean subperiosteal resection had no effect upon the symptoms; subsequent resection of the periosteum gave complete relief.

Gentleness of manipulation, clean dissection, and a bloodless operative field are essential, as well as an accurate knowledge of the anatomical relations of the parts concerned. The importance of the last-named point is well brought out by Coote [4] in the somewhat quaint account of the first recorded case of an operation for cervical rib which I have been able to find.

Results of Operation.

In my series there has been no fatality, and in no instance has any wound infection occurred. In only two instances did any ill-effect follow the operation. In one a severe organic monoplegia developed, which only cleared up after many months. In another a hysterical paralysis occurred, for which treatment was refused, and the patient was lost sight of.

The results are most fairly presented in the brief notes to be found in the tables, for each of the most prominent group of symptoms deserves separate notice. These tables were prepared before the War, and it has been interesting to note that, in reviewing the cases after a further long period, some which were previously entered as "relieved" have had to be transferred to the "cured" column, whilst others, where relapse has occurred, have had to be transferred to the group marked "relieved."

Pain.

As regards pain, twenty-seven patients have been followed for a sufficiently long period to judge of the result; nineteen of them are cured and the rest very considerably relieved. Where pain is the predominant symptom it may safely be predicted that the result of operation will be gratifying, and that the relief of pain will usually be immediate and permanent.

Result of Operation as regards Pain.

Cured.

Case 2.—Female, aged 38, housekeeper. History five years. Constant aching along ulnar side of whole arm and shoulder. After eight months, pain only in opposite arm. After seven years, "pain gone but fingers drawn."

Case 5.—Female, aged 51, housework. History three years. Pain in right shoulder and occasional neuralgic pain along ulnar border of forearm and hand; also wasting and vasomotor symptoms. Immediate relief of pain. Ten months later no pain, but still some weakness. Seven years later, remains free from pain; "constantly using my hands from 7 a.m. to 9 p.m."

Case 7.—Female, aged 36, housework. History five months. Neuralgic pain in shoulder and inner side of arm, forearm and hand; also weakness and wasting. Immediate relief of all pain. Remains well and at work six months later. Seven years later, "occasional pain in both arms."

Case 8.—Female, aged 50, housework. *Left side:* History six months. Neuralgic pain in shoulder, inner side of arm, forearm and hand, increased by work; also weakness. Ten months later no pain. Occupation unchanged. *Right side:* Symptoms exactly similar to those of left side. Immediate relief. After eight months, completely free from pain. After six years, right completely free; left free until two years ago, when occasional pain after unusual exertion.

Case 11.—Female, aged 52, housework. History three years. Aching in shoulder and neuralgic pain in thumb. Immediate relief of pain. Remains free from pain after ten years.

Case 16.—Female, aged 22, clerk. *Left side:* History two years. Pain in shoulder, inner side of elbow and inner fingers. Immediate and complete relief of pain. Remains well five years later. No change of occupation, *Right side:* History four months. Pain in shoulder, inner side of forearm and wrist. Immediate relief of pain. Remains free five years later.

Case 21.—Female, aged 25, housework. History two and a half years. Also vasomotor symptoms. Aching pain from shoulder to fingers—worst on inner side of forearm. Immediate and complete relief of pain. Lost sight of.

Case 24.—Female, aged 38, cook. History three years. Aching pain in shoulder and arm, also wasting. Immediate relief of pain. Remained well and at work nine months later. Lost sight of.

Case 27.—Male, aged 12, school. History four months; also wasting. Aching pain in ulnar border of forearm and hand. Immediate relief of pain. Remained well eighteen months later.

Case 30.—Female, aged 30, barmaid. History seven years. Pain in elbow and hand; also vasomotor symptoms and weakness. Immediate relief of pain

Six months later only occasional twinges. Six years later, "pain entirely gone."

Case 31.—Female, aged 15. History one month. Pain in shoulder and outer border of arm and forearm ; also weakness. Immediate relief of pain. Remains well six years later.

Case 34.—Female, aged 42, secretary. History six weeks. Aching along ulnar border of arm ; also wasting and vasomotor symptoms. Immediate relief of pain. Free from pain seven years later.

Case 37.—Female, aged 27, needlework. History two years. Aching pain in shoulder and arm ; also vasomotor symptoms. Immediate relief of pain. Remains well seven years later. No change of occupation.

Case 38.—Female, aged 33, cook. History four years. Pain in shoulder and hand, also weakness ; immediate relief of pain. Remains well three months later. Lost sight of.

Case 40.—Female, aged 42. History six years. Aching in shoulder and arm, also vasomotor symptoms and wasting ; immediate relief. Still completely free from pain six years later. "Life worth living."

Case 42.—Male, aged 45, medical practitioner. History eight years. Very severe neuralgic pain in shoulder and arm generally, but chiefly along middle of forearm and outer border of hand. Immediate and complete relief of pain. Remains well and in full work eight years later. "Never any pain since I recovered from the anæsthetic. The operation altered my whole life."

Case 43.—Female, aged 34, housework. History one month. Pain in arm generally ; also wasting. Immediate relief of pain. Seven years later, "operation completely successful."

Case 46.—Female, aged 53, housework. History seven months. Pain in right arm. Immediate relief of pain. Remains well eight years later.

Case 49.—Female, aged 18, cook. History two and a half years. Neuralgic pain in outer side of arm and hand. Immediate relief of pain. Remained well three months. Recurrence. Second operation for excision of scar tissue in track of previous subperiosteal resection. Complete relief, and remained well two years later. Subsequently lost sight of.

Relieved.

Case 12.—Female, aged 49, housework. History ten years. Aching pain in thumb ; more recently pain in shoulder and arm, also wasting. Two months later pain gone. Eight years later reports herself much better.

Case 13.—Female, aged 38, letter-sorter. *Right side :* History fourteen years. Diffuse pain in arm, also weakness. Immediate relief of pain. Four months later free from pain. Three years later only has pain occasionally. Nine years after operation writes : "Pain nearly gone ; I only get attacks. I am getting back all the power and strength again, and that after fourteen years of intense suffering and nearly lost the use of my arms and hands."

Left side : Condition similar. Nine months later still some pain. Eighteen months later has pain occasionally.

Case 15.—Male, aged 42, painter. History six years. Pain in neck, outer side of arm and back and outer side of forearm ; also weakness. Immediate relief of pain. Two years later feels some pain after day's work. Subsequently lost sight of.

Case 22.—Female, aged 42, housework. History five years. Pain in elbow and fingers ; also weakness. Complete relief of pain. Patient lost sight of.

Case 23.—Female, aged 24, school teacher. History six months. Pain in arms and neck, also wasting. Pain relieved at once. Eleven years later " sometimes a deal of pain at times."

Case 26.—Female, aged 50, housework. History 4 years. Diffuse pain in arm ; also vasomotor symptoms and wasting. Eighteen months later very much better, but exertion brings on pain occasionally.

Case 35.—Female, aged 53, nurse. *Left side :* History two years. Pain in arm, chiefly outer border and flexor surface. Three years later much improved but gets pain after exertion. Ten years after operation " pain only after excessive work." *Right side :* Condition exactly similar.

Case 48.—Female, aged 19, clerk. History twelve months. Aching pain from elbow to wrist; also weakness. Immediate relief of pain. Eighteen months later, occasionally gets pain in cold weather. Subsequently lost sight of.

VASOMOTOR SYMPTOMS.

In many cases the relief of the paræsthesiæ and other symptoms which I have grouped as " vasomotor " is immediate. Others however evidently require a considerable time, amounting to many months or even years, before recovery is complete or even approaches completeness. Thus Case 6 was noted, sixteen months after operation, as " remaining much the same as before," but seven years later the report is " no trouble of any kind for some years."

RESULT OF OPERATION UPON THE VASOMOTOR SYMPTOMS :

Namely—Objective (swelling, coldness, blueness).
Subjective (acroparæsthesia).

Cured.

Case 1.—Female, aged 13, school. History twelve months. Almost pure vasomotor case. In six months little result ; in eighteen months cured. " No trouble of any kind."

Case 3.—Male, aged 27, stoker. History six months. Almost pure vasomotor case. Much immediate improvement—practically well in sixteen months. No change of occupation. Eight years later—" While in France

and under bad conditions I felt it several times but never bad enough to go sick with. Since my return I have not had the slightest trouble."

Case 4.—Female, aged 18, domestic servant. History three years. Also weakness, but symptoms chiefly vasomotor. Better immediately ; still improving six months later ; practically well in twelve months. No change of occupation.

Case 5.—Female, aged 51, housework. History three years. Also wasting. Better and still improving ten months later. Eight years after operation, no vasomotor symptoms.

Case 6.—Female, aged 50, housework. History eight months. Remains much as before sixteen months later. Eight years after operation, "no trouble of any kind for some years."

Case 10.—Female, aged 26, dressmaker. History eighteen months. Also wasting. Subjective sensory phenomena disappeared at once. No change of occupation. Ten years after operation, "no symptoms whatever."

Case 11.—Female, aged 52, housework. History three years. Almost pure vasomotor case. Relieved immediately ; almost well in a month ; after fourteen months had been perfectly well for some months ; after three years remains perfectly well. No change of occupation. Ten years after operation, "just occasionally a little of the old feeling of numbness and pins and needles in the hand which occurs at night after doing more work than usual."

Case 14.—Female, aged 30, nurse. History five years. Also pain. Relieved immediately and still improving four months later. Eight years after operation, quite well.

Case 21.—Female, aged 25, no occupation. History two and a half years. Almost pure vasomotor case. Immediate relief. Patient lost sight of after two months.

Case 37.—Female, aged 27, fur sewer. History eight months. Also pain. Immediate disappearance of vasomotor phenomena ; remains well eighteen months later. No change of occupation. Eight years after operation, "I have had no further trouble with my arm."

Case 38.—Female, aged 33, cook. History four years. Also wasting and weakness. Immediate relief of vasomotor symptoms. Three months later, "use of hand quite restored."

Case 39.—Female, aged 41, telegraphist. History two years. Almost pure vasomotor case. Some wasting. Immediate relief. Two months later, still improving. Fifteen months later, cured for some months and remains so.

Case 40.—Female aged 42. History six years. Also wasting. Vasomotor symptoms gone in a week. At work and remains cured six years later.

Case 42.—Male, aged 45, M.D. History eight years. Also pain and weakness. Immediate and complete relief of all symptoms. Remains well and in full work ten years later.

Relieved.

Case 18.—Female, aged 31, needlework. History eighteen months. Also pain and some wasting. Immediate relief. Two years later still improving, but symptoms recur in modified form after exertion. "Hand sometimes feels hot and tingling at night after exertion. Feels the cold greatly."

Case 25.—Female, aged 53, lady's maid. History ten years. Chiefly vasomotor case; but also weakness and wasting. Immediate relief; resumed work; very much better but not quite well three and a half years later. Ten years after operation, "left arm and hand painful when I have been doing too much. Otherwise I do not feel anything of it."

Case 26.—Female, aged 50, housework. History four years. Also, and chiefly, pain and weakness. Vasomotor symptoms improved at once: still improving after six months; in two years still better but not quite well. Heavy work brings on pain.

Case 30.—Female, aged 30, barmaid. History seven years. Chiefly vasomotor; also weakness and wasting. Improved at once; six months later better still and at work. Seven years after operation, "pain entirely gone, but in cold weather hand sometimes becomes useless."

Case 34.—Female, aged 42, secretary. History three months. Also pain and wasting. Vasomotor symptoms disappeared at once. Seven years later, "cold quickly stiffens the fingers."

Case 50.—Female, aged 49, housework. History twelve years. Chiefly vasomotor case at first; later wasting. Immediate relief of vasomotor symptoms. Patient lost sight of.

Unrelieved.

Case 19.—Female, aged 42, housework. History eighteen years. Also wasting. No improvement in six months. Lost sight of.

Case 41.—Female, aged 26, housework. History ten months. Only slight vasomotor symptoms; chiefly pain and wasting. No improvement in eight months. Lost sight of.

Muscular Wasting.

During the Discussion on Cervical Ribs at the Royal Society of Medicine in 1913, Thorburn stated that he had never been able to satisfy himself that atrophy of the hand muscles was ever entirely cured. This opinion was based upon an experience of twenty cases, observed over periods varying from twelve months to fourteen years. This was an important statement which, coming from such a source, could not be lightly passed over, and I have therefore reviewed my own cases with particular regard to this point. In thirty-one out of fifty cases wasting of the hand muscles was a pronounced feature. Twelve may be regarded as completely recovered both as regards bulk of

muscle and strength. In twelve cases the improvement was quite definite, but incomplete; whilst in only seven did any improvement at all fail to appear. It should be noted, however, that four of these seven patients were lost sight of before the result could fairly be judged. As might be expected, the recovery of the affected muscles takes a long time, and doubtless this is influenced by the treatment bestowed after operation. It is impossible to say what influence this factor may have had in my cases. As may be observed from the tabulated results it is on the whole the cases with the shortest histories which have done best as regards the muscular wasting.

Result of Operation on the Wasting of Muscles.

Cured.

Case 1.—Female, aged 13, school. History twelve months. General slight wasting. After six months no wasting to be detected. After eighteen months "no trouble of any kind; appears completely cured."

Case 6.—Female, aged 50, housework. History eight months. All intrinsics wasted. In sixteen months, little change; in eight years hand muscles quite filled out.

Case 8.—Female, aged 50, housework. History six months. Slight wasting of all intrinsics. After ten months, muscles filling out. After eight years muscles quite normal.

Case 10.—Female, aged 26, dressmaker. History two years. Marked wasting of thenar muscles. After two years muscles improving; in another six months quite filled out. After seven years remains completely well. No change of occupation.

Case 18.—Female, aged 31, housework. History eighteen months. Slight wasting of all intrinsics. After twelve months, "muscles increasing; hand stronger." After seven years remains much the same.

Case 23.—Female, aged 24, school teacher. History six months. Wasting of all intrinsics. After four years muscles much improved. After eleven years muscles full bulk and power.

Case 25.—Female, aged 53, lady's maid. History three months. Wasting chiefly of thenar muscles. After two years "muscles improving;" after three years "full bulk." After ten years remains well.

Case 27.—Male, aged 12, school. History three months. Marked wasting of all intrinsics. After eighteen months, full use of hand.

Case 33.—Female, aged 41, housework. History seven months. All intrinsics wasted. After twelve months marked improvement in bulk of muscle. After eighteen months "complete recovery." Seven years later "entirely recovered."

Case 38.—Female, aged 33, cook. History four years. Some wasting of all intrinsics. After two months visible improvement. After twelve months, complete recover .

Case 39.—Female, aged 41, telegraphist. History two years. All intrinsics wasted. After three years, complete recovery.

Case 40.—Female, aged 42. History five months. Wasting: chiefly thenar. After eight months muscles much filled out. After five years, completely recovered.

Relieved.

Case 2.—Female, aged 38, housekeeper. History 5 years. All intrinsics, but specially opponens and abductor. After six months, no change. After seven years, "pain gone but fingers drawn."

Case 4.—Female, aged 18, domestic servant. History three years. Chiefly opponens and abductor. After twelve months, much improvement.

Case 7.—Female, aged 36, housework. History four months. All intrinsics but chiefly of thumb. After five months, much improved. Seven years later condition much the same.

Case 11.—Female, aged 49, housework. History twelve months. Wasting of abductor pollicis. After five years, "ever so much better." After ten years, remains practically well.

Case 20.—Female, aged 27, nurse. History fifteen months. Wasting of all intrinsics. After two years, muscles better; hand stronger. Not traced further.

Case 24.—Female, aged 38, cook. History three years. Wasting of all intrinsics. After nine months, very great improvement. Lost sight of.

Case 26.—Female, aged 50, housework. History four years. Wasting of all intrinsics. After eight months, muscles filling out. Not traced further.

Case 29.—Female, aged 18, housework. History two years. All intrinsics wasted, but chiefly thenar. After eighteen months, muscles fuller but still less than left. After nine years, no symptoms, but no further recovery in bulk of muscle.

Case 30.—Female, aged 30, clerical. History seven years. Wasting of all intrinsics. After six months, practically no change. Seven years later, "pain entirely gone. Strength of hand not completely restored."

Case 44.—Female, aged 52, needlework. History eighteen months. All intrinsics wasted. After three years, bulk of muscles much improved.

Case 47.—Male, aged 62, painter. History two years. After two years slight improvement in bulk of muscles.

Case 48.—Female, aged 19, domestic service. History twelve months, Wasting chiefly thenar. Eighteen months later, some improvement.

Unrelieved.

Case 5.—Female, aged 51, housework. History three years. Chiefly opponens and abductor. After nine months, stronger but muscles have not filled out. Seven years later, "condition much the same."

Case 17.—Female, aged 38, housework. History three years. Profound wasting of thenar eminence; slight of other intrinsics. After six months. "I fancy my hand is improved, but it is very slight." After seven years remains much the same.

Case 19.—Female, aged 43, housework. History five months. Wasting of thenar muscles. After six months, no improvement. Lost sight of.

Case 34.—Female, aged 42, secretary. History six weeks. All intrinsics wasted, but chiefly thenar. After seven years, no improvement but no worse.

Case 41.—Female, aged 26, housework. History ten months. All intrinsics wasted. After eight months, no improvement. Lost sight of.

Case 45.—Female, aged 53, married. History eight years. All intrinsics wasted. After two months, no change. Lost sight of.

Case 50.—Female, aged 49, housework. History twelve years. All intrinsics greatly wasted. In thenar muscles no response to faradism. Not traced.

Summary.

(1) Variations in the composition of the brachial plexus are apt to be associated with costal abnormalities, prefixation with a seventh cervical rib and postfixation with an abnormal first thoracic rib. There is, however, no regular relationship between the costal and neural anomalies.

(2) Of the several different types of cervical rib met with clinically, that which most frequently requires treatment by operation is represented by an abnormally large non-jointed costal process, continued onwards as a dense fibrous band, to be attached to the first thoracic rib behind the sulcus nervi brachialis (sulcus subclaviæ).

(3) Symptoms of gradual onset result from continual slight traumatism to the eighth cervical root or lowest cord of the plexus, caused by the tightening of the band during respiration and in certain movements of the arm.

(4) With a postfixed plexus symptoms referable to the first thoracic root may be caused by the pressure of a normal first thoracic rib.

(5) "Vascular symptoms" are vasomotor in origin and result from injury to the sympathetic fibres, shortly after their entrance into the eighth cervical and first thoracic roots.

The subclavian artery, whilst often occupying an abnormally high position in the neck, nevertheless rarely lies upon a bony cervical rib. It may be just held off from the first thoracic rib by the fibrous band.

(6) The results of operative treatment in fifty cases are given, the majority having been traced for a period of from two to twelve years.

Pain was cured in nineteen cases, and relieved in eight.

Muscular wasting was cured in twelve cases, relieved in twelve, and unrelieved in seven.

Vasomotor symptoms were cured in fourteen cases, relieved in six, and unrelieved in two.

EXPLANATION OF THE DIAGRAMS.

The diagrams have been constructed from rough sketches made at the time of operation. Those parts of the drawings which represent the abnormal ribs were made from the actual specimens removed, their relations to the other parts being shown in a diagrammatic manner. The dissections of a normal plexus (1) and of a plexus associated with a rudimentary thoracic rib (XVI) have been reduced to the same semi-diagrammatic form for the sake of comparison. The relative size of the nerve trunks as indicated in the diagrams must be taken as only approximately correct. A contribution from T1 is represented in all cases except those in which it seemed quite certainly absent.

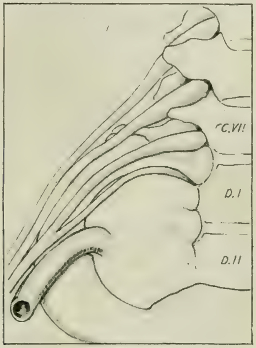


FIG. 4.—From a dissection of a normal plexus, showing the very small costal process of C7 vertebra, and the relation of the nerves with it and T1 rib.

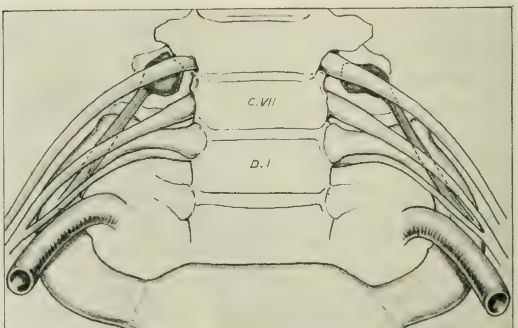
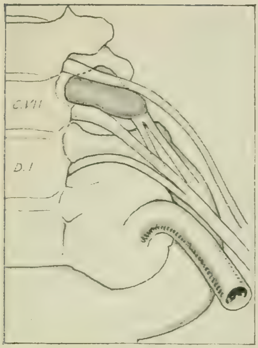


FIG. 5 is an example of Type 1, which radiographically was deemed to have no cervical rib. The report was "well marked transverse processes." This patient remains well eleven years after the bilateral operation.



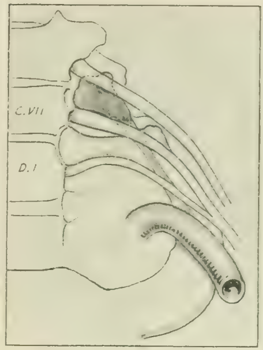
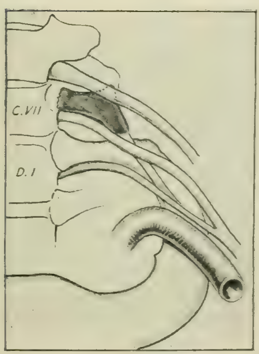
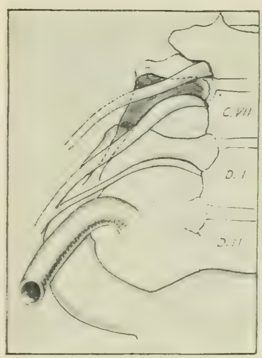


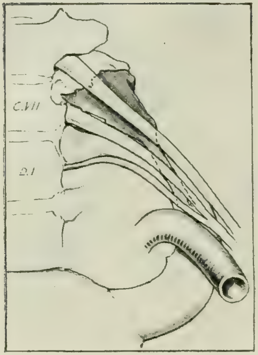
FIG. 6 represents a similar rib to those shown in fig. 3, but the rib is longer and was more pointed forwards, so that the band was more prominent. This patient remains well ten years after operation.

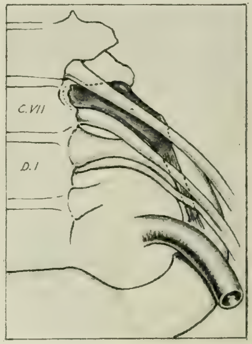
FIG. 7 is an example of Type 1, and may be taken as representing what in my experience is the commonest type which causes symptoms.



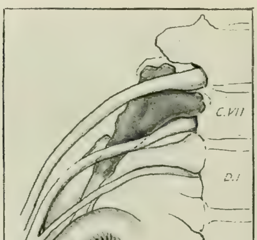


FIGS. 8 and 9 are examples of Type 2. The ribs are well formed at their proximal part, and the joints are perfect, but almost the whole body is represented by non-ossified tissue.





FIGS. 10 and 11 are examples of Type 2 merging into Type 3. The very sharp edged bone which separates C7 and C8 in fig. 10 is represented by a more rounded ridge in fig. 11.



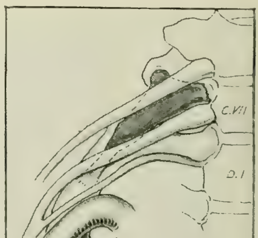


FIG. 12 is an example of Type 3, where the rib is long enough to carry C8 upon its bony portion.

FIG. 13 belongs to Type 3 and approximates to Type 4.

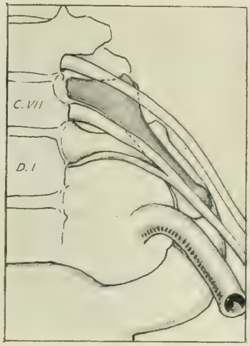
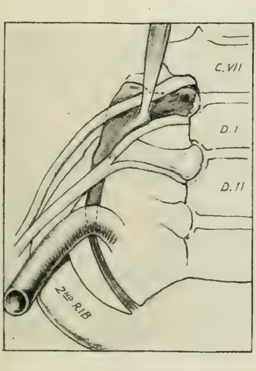


FIG. 14 from a specimen in the Museum of the Royal College of Surgeons (No. 505) showing a rib intermediate in character between Types 3 and 4.

FIG. 15 is an instance of Type 4, there being a joint between the enlarged end of the cervical rib and an irregular mass of bone on the upper surface of T1 rib.



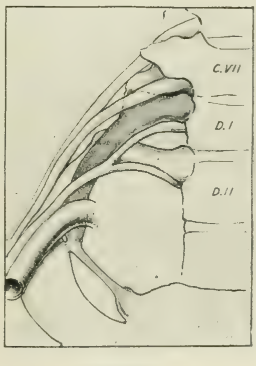


FIG. 16 is the only instance of Type 5 in my series.

FIG. 17 is constructed from a dissected post-mortem specimen shown to me by Prof. Wood Jones (10) and reported by him.

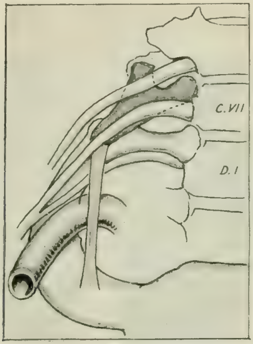


FIG. 18 represents a unique specimen which does not conform to any of the Types described. (See text.)

REFERENCES.

[1] BRAMWELL, E. *Rev. Neurol. and Psychiat.*, 1903, **1**, 236.
[2] *Idem* and J. DYKES. *Edin. Med. Journ.*, 1921, **27**, 65.
[3] BUZZARD, E. F. *Brain*, 1902, **25**, 299a.
[4] COOTE. *Lancet*, 1861, **1**, 360.
[5] DWIGHT. Quoted by WOOD JONES, *Journ. Anat. and Physiol.*, 1887, **21**, 539.
[6] HARRIS, WILFRED. *Journ. Anat. and Physiol.*, 1904, **38**, 399.
[7] HERTSLET and KEITH. *Journ. Anat. and Physiol.*, 1896, **30**, 562.
[8] HOWELL, C. M. HINDS. *Lancet*, 1907, **1**, 1702.
[9] JONES, F. WOOD. *Journ. Anat. and Physiol.*, 1910, **44**, 377.
[10] *Idem.* *Journ. Anat. and Physiol.*, 1911, **45**, 249.
[11] JONES, H. LEWIS. *Quart. Journ. of Med.*, 1907-8, **1**, 187.
[12] KEEN, W. W. *Amer. Journ. Med. Sci.*, 1907, **133**, 173.
[13] LANE, W. ARBUTHNOT. *Journ. Anat. and Physiol.*, 1885, **19**, 266.
[14] LUCAS, M. F. *Journ. Anat. and Physiol.*, 1915, **49**, 336.
[15] MORLEY, JOHN. *Clin. Journ.*, 1913, **42**, 461.
[16] MURPHY, THOMAS. *Austral. Med. Journ.*, 1910, **15**, 582.
[17] PAGET, Sir J. "Clinical Lectures," ed. 2, Lond. 1879.
[18] STOPFORD and TELFORD. *Brit. Journ. Surg.*, 1919-20, **7**, 168.
[19] THOMAS and CUSHING. *Johns Hopkins Hosp. Bull.*, 1903, **14**, 315.
[20] THOMPSON, THEODORE. *Brain*, 1908, **31**, 298.
[21] THORBURN, WILLIAM. *Med. Chronicle*, 1907-8, **47**, 165.
[22] TODD, T. W. *Journ. Anat. and Physiol.*, 1912, **46**, 244.
[23] *Idem.* *Lancet*, 1912, **1**, 362.
[24] WILSON, S. A. K. *Proc. Roy. Soc. Med.*, 1913, **6** (Clin. Sect.), 133.

ON STRUCTURAL LAWS IN THE NERVOUS SYSTEM: THE PRINCIPLES OF NEUROBIOTAXIS.[1]

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Amsterdam.

THE factors that determine the structures in the nervous system, the form of the neurones, their position and connections, have puzzled the minds of many neurologists.

This matter was first considered more thoroughly by embryologists, who, however, were chiefly interested in the problem of the connections between the central nervous system and the peripheral organs, the formation of nerve-roots.

One of those was Hensen, who assumed "that all nerves originated by an insufficient separation in the *primary* connections between ganglion cells and their peripheral organs during evolution." Hensen was thus a forerunner of the conception that a real separation of the cellular constituents of the body never occurs, but that its elements remain connected with each other. A similar conception of *syndesmism* was defended in England by Sedgwick, who stated that "Nerves are developments of the reticulum, elongated strands of the pale substance composing this reticulum, with some of their nuclei."

I shall come back later to this conception, which, although it may contain a true principle, can never explain the peculiar selectivity in the neuronal connections.

An opposite opinion was held by Balfour and His, who showed that the connections of the nerves with their end-organs were secondarily acquired.

Of these two leading embryologists, His was generally inclined to regard mechanical factors as the most important in the embryonic evolution, and he practically saw in the growth and arrangement of the nervous elements a purely mechanical problem.

He attempted to solve this problem by assuming that the direction of the nervous offshoots was determined by the places or paths of least resistance.

The Belgian histologist Dustin[2] has pointed out that by such *preformed forepaths* we are able to understand why the *regeneration* after cutting a peripheral nerve runs so smoothly, guided by the degenerated nerve-sheaths of the peripheral stump; whereas in the central nervous system, which does not exhibit any sheaths of Schwann, regenerating nerve-fibres rarely reach

[1] A lecture read in the University of London.

[2] Who termed this the hodogenetic principle.

their functional end-point, and thus the regeneration is mostly without result. Although this may be true, the presence of eventual forepaths is not sufficient to explain the *formation* of nerve-fibres and besides only displaces the solution of the problem.

If it be assumed that only a general tendency for making offshoots is present in the ganglion-cell, and that the *direction* in which the offshoots shall go only depends upon previously existing paths in the surrounding tissue, then this question remains, namely, by what influence is such a typical arrangement of the surrounding tissue established, so that just the *functionally correct* nervous connections result from it?

Especially the exquisite functional character of the connections in the central nervous system, where such conducting sheath-cells do not occur, remains entirely unexplained by this theory.

Another embryologist, Held, proceeds from the correct assumption, already formed by Hensen and Sedgwick, that the nerve-cells are *ab origine* connected by intracellular bridges, plasmodesms, previous to the formation of fibrils. Held was actually able to demonstrate that later on the fibrils (the nerve-tracts) grow into such plasmodesms. Held himself, however, is perfectly convinced of the fact that the general presence, the ubiquity, of these plasmodesms can never explain the selection in the formation of the fibrils. It can never explain why nerve fibrils grow in some plasmodesms and not in others.

He believes that the topography of the cells ([34], p. 47) and the direction of their axes ([34], p. 68) may act a part, but Held himself does not believe that these factors are sufficient to explain the typical *selection* in the formation of nervous tracts and says ([34], p. 270) that the determining principle has as yet escaped our scientific research.

A similar remark is made by Harrison, to whom we owe those valuable researches on the outgrowth of nerve-fibres in cultures, and who stated himself "that there is nothing in this work which throws any light upon the process by which the final connection of a nerve-fibre is established." That it must be a sort of specific reaction between each kind of nerve-fibre and its particular end-point is clear, according to Harrison.

The first who supposed a tropistic influence to act a part in these structures and who also accepted a reciprocal tropistic influence of the central nervous elements on each other was Cajal. The work accomplished by this author is extremely valuable.

Though—as is admitted by Cajal himself—it could not solve the problem, my respect for this excellent Spanish neurologist does not allow me to proceed without mentioning his theory.

Cajal believes that the connections of the nervous elements is determined by the secretion of *attracting and repulsing substances* and by the sensibility to these substances in the ganglion-cells.

Concerning the secretion of these chemotactic substances, which,

according to Cajal, may also be secreted by the ependyma cells,[1] this writer assumes that different parts of the central nervous system enter this stage of secretion at different periods of their embryonic development.

Cajal says that the stage of attraction *coincides with the evolution of the cell.* This, however, is apparently a displacement of the difficulty, for Cajal does not make any statement as to which factors determine the evolution of the cells and consequently of the secretion of those substances.

Thus he does not give an explanation concerning the cause of the local and temporary selectivity in this chemical process nor of the factors that determine the different nature of the axons and dendrites (the so-called *dynamic polarization of the neuron*).

Cajal himself remarks in his work on the retina: "Cette théorie présuppose des conditions préalables chimiques et morphologiques tout à fait inexplicables : on peut dire que cette théorie éloigne la difficulté sans cependant parvenir à la résoudre"[2] ([67], p. 240).

More important than Cajal's chemotactic theory is a remark by this author concerning the shifting of nerve-cells in the embryonic development of the nervous system, of greater importance even than he himself has realized.

This remark (not known to me when I commenced my researches on this question) is in perfect harmony with my own observations, and I am the more glad to be able to confirm Cajal's observations as to this point, since I arrived at a similar result in an entirely different way, namely, not by embryonic, but by comparative phylogenetic studies.

The remark then made by Cajal runs as follows: "If during embryonic development new axons pass to some region of the central nervous system, ganglion-cells may approach these axons in two different ways, either by sending forth long dendrites, or by a migration of the cell-body itself" ([69], p. 560).

Cajal mentions, as examples of a shifting of cells, the superficial layer of granular nerve-cells which in an early period of development

[1] Although I am not inclined to attribute an extensive part in this to the central spongioblasts it is a fact that different non-nervous elements (lemnoblasts, muscular cells, epithelium and even cicatrices of the connective tissue) are able in certain stages of their development to direct the offshoots of nervous elements.

[2] Child also ([17], p. 170) has recently criticized the chemotactic theory, and appears, like myself, to be an adherent of the bio-electric theory (see at the end of this paper).

cover the surface of the cerebellum and which later on shift into the depth of the cerebellum.

He also mentions the cells of the spinal ganglia which originally lie next to the neural tube, but later on shift a short distance peripherally away from the spinal cord.

Comparative researches on the oblongata and the mesencephalon have convinced me that this shifting of nerve-cells is a very common appearance, and that these shiftings of nerve-cells are determined by a process of *taxis* or *tropism* that is caused by the stimulation of such cells and their bio-electric consequences, which determine also the selectivity in the neuronal connections, and the difference between the dendrites and axons, the so-called *dynamic polarization of the neuron.*

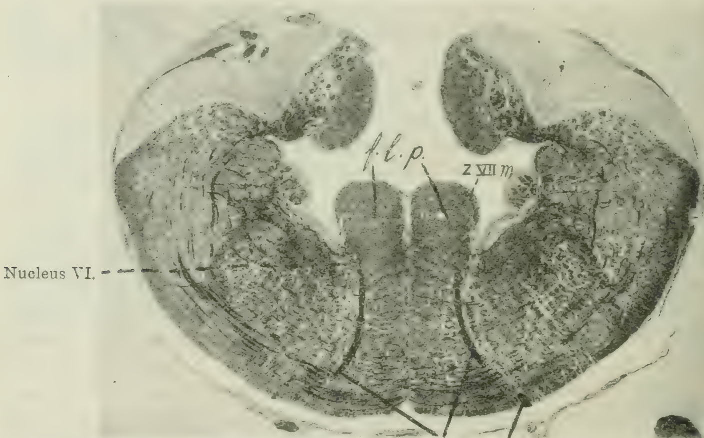

FIG. 1, A.—Dorsal position of the abducens nucleus in a shark (Acanthias) in which the dorsal longitudinal bundle is very large. After Van der Horst.

In what manner these processes can be engrammatically reproduced under embryologic conditions cannot for the present be stated. The same applies to the entire ontogeny however. Also the formation of the extremities for walking and grasping can only be explained by engrammatic factors, the character of which escapes us up to now. It may be that the electric potentials arising during evolution (Child) and whose sequence may be determined by engrammatic factors act a part in this process.

During the past twelve years I have registered the topographic differences of homologous cell-groups in the central nervous system of vertebrates where such differences appear most clearly in the nuclei of the motor roots of the oblongata (cf. Black and v. d. Horst), but also in many cells of the spinal cord, the mid- and forebrain (Herrick, Elliot Smith, Dart).

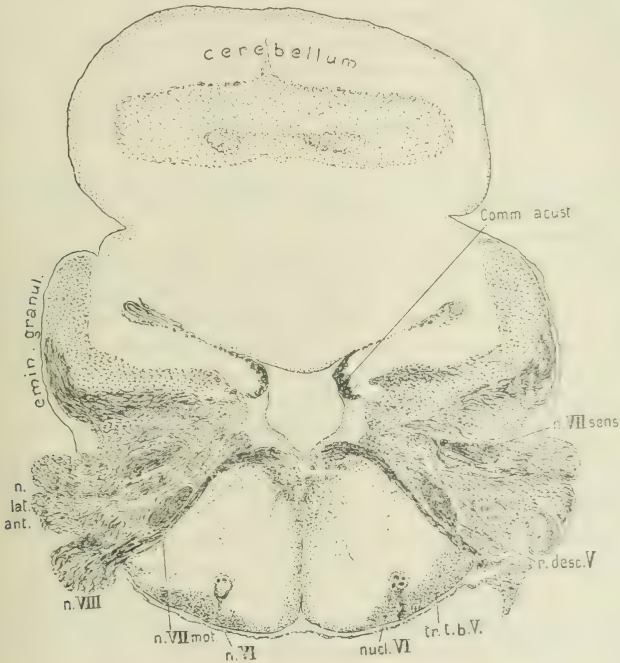

FIG. 1, B.—Ventral position of the VIth nucleus in a bony fish (Mugil chelo) with small dorsal longitudinal bundle and large ventral reflex tract (tr. t. b. V.). After Van der Horst.

As it is clear that the topography of these cell-groups is determined by the places from which the largest number of excitations reach the cells, we have plainly to do with an appearance of taxis or tropism, which I have termed *neurobiotaxis*, since it occurs in the nervous system during life.

For a better insight into this process of neurobiotaxis let us consider

figs. 1, A and 1, B, where the dorsal position of the abducens nucleus in a shark, with its large dorsal longitudinal bundle, strongly contrasts with the ventral position of the same nucleus in a bony fish where the dorsal longitudinal bundle is small, but where the ventral reflex-tracts, that influence this cell-group, develop to a much greater size (the tr. tecto-bulbaris ventralis : tr. t. b.).

In these cases it is evident that with an increase of stimulations in a certain tract not all the cells of the bulb approach this tract, but only certain cells, namely, such cells as bear a certain relation to this tract; whereas other cells, for instance, the cells of the facial nucleus that innervates the gills, do not migrate in the direction of this strengthened reflex field, since their function is not related to the function of these tracts.

So we see that there is a *selection* and that obviously a certain relationship is required for this process of shifting. We further see that this relationship is a functional one and that it consists in a simultaneous excitation of the enlarged stimulation centre and the motor-cells. If the motor-cells have a stimulative relationship with the enlarged centre, that is, if the motor-cells are in action at the same moment in which a nervous current passes through the enlarged tract, as is the case with the eye-muscle nuclei and optic reflex tracts, then the cells are attracted by those tracts, not otherwise.

This is anatomically expressed by the fact that the nucleus of the abducens shifts from one path of visual reflexes (the dorsal longitudinal bundle) to another region of visual reflexes (the tractus tecto-bulbaris ventralis). An increase of the central *taste fibres* of the oblongata however has not the slightest influence upon the position of the eye-muscle nuclei, but causes migrations of the nuclei of the jaws and gills.

It is thus seen that the relation of the (dendrites and) ganglion-cells in the nervous system is regulated in conformity with that law which in psychology has long been known as the *law of association*, in which law (in all forms in which it may appear) the simultaneity or direct successivity of the excitation is the chief point.

This observation, made with motor-cells and their dendrites, led me to study more fully the course of the axons, and I found that a critical observation of the relations existing between the regions where they begin and end show the same law, namely, that only a functional relationship between two regions can cause an axonal connection between those regions. As a simple example it may be mentioned that axonic connections may arise and do in fact arise between the visual centres of the brain

and the vestibular centres on account of the fact that visual impressions and vestibular impressions very often reach the brain simultaneously.

Thus, *associated stimulation* was found to be the determining factor in the neurotropic appearances, and I was able to formulate the facts in the following words :—

(1) *If several stimulative charges occur in the nervous system, the outgrowing of the chief dendrites and eventually the shifting of cells takes place in that direction whence the largest number of stimulations goes to the cell.*

(2) *This outgrowing or shifting, however, only takes place between stimulatively correlated centres ; temporarily correlated excitation acts a part also in the connections of the axons.*

FIG. 2 shows that the outgrowth of the dendrites and the final shifting of the cell body itself takes place in a stimulo-petal direction, whereas the course of the axis-cylinder is stimulo-concurrent.

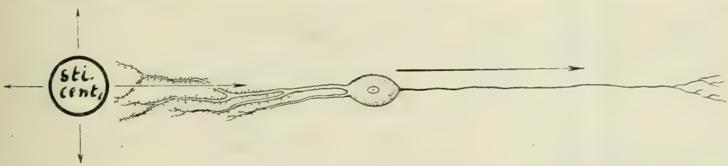

FIG. 2, A.—Giant dendrites grow out in the direction of the centre from which the stimuli proceed (sti. cent.).

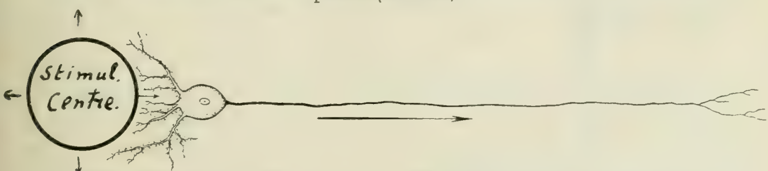

FIG. 2, B.—The cell body itself (perikaryon) has shifted in the direction of the stimulation centre. The dendrites have shortened, the axis-cylinder has correspondingly enlarged.

Comparative anatomy of the fibre-tracts in the central nervous system gives plenty of evidence that this is always a leading principle in the formation of tracts. Now, however, the question arises : if associated excitation causes the formation of dendrites and the shifting of ganglion-cells as well as the formation of axons, how may we then explain the different character of the dendrites and the axon, the so-called *dynamic polarization* of the neuron ?

Whereas the approaching of the dendrites and nerve-cells towards

the centre of excitation is a stimulo-petal tropism, that is, a tropism that runs to the centre of stimulation, the problem with the axon is much more difficult since the axon apparently does not grow out towards the excitation centre, but in the same direction as the nervous current that irradiates from this centre, and consequently grows away from the excitation centre (fig. 2).

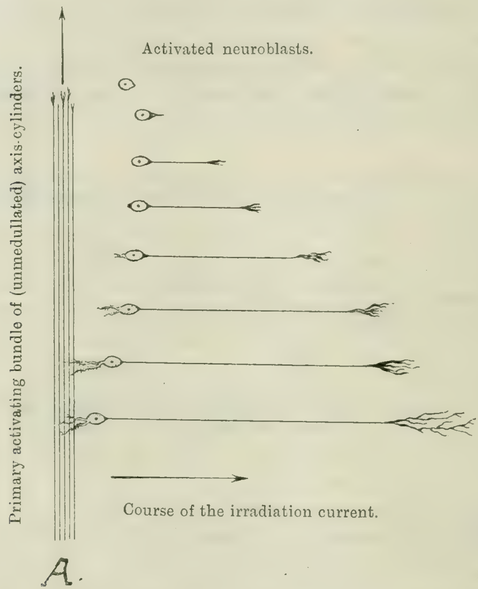

FIG. 3.—The activation of adjacent neuroblasts by a non-myelinated growing bundle of nerve-fibres. The vertical arrow indicates the direction of the growth and the stimulation current in the primary activating bundle which has started at A. The horizontal arrow indicates the direction of the irradiating influence, perpendicular to the primary bundle. The proximal cells are sooner and consequently further differentiated than the distal cells. After Bok (slightly modified).

That the axon actually does grow out in the same direction as the excitation current, and that this current plays an important part at its formation, has been proved by my former scholar, Bok (1915).

Bok found that when a bundle of unmedullated nerve-fibres grows out and passes neuroblasts on its way, these neuroblasts become activated by that bundle and send forth an axon in a direction perpendicular to

the activating bundle, apparently growing in the same direction as is taken by the current that irradiates sideways from the growing bundle (see fig. 3).

This fact was established in a manner that does not admit of any doubt, since it was found that the neuroblasts lying in the region of the growing bundle were only activated if the activating bundle reached their level, apparently by stimuli irradiating therefrom.

Thus here, too, we find a process that, like the outgrowth of the dendrites and the shifting of the cells, is determined by a nervous current.

At the same time, however, we perceive that the axon (in contrast to the dendrites and the shifting of the cell body) grows away from the region of stimulation.

Whereas the growth of the dendrites is a stimulo-petal tropism, the growth of the axon bears a *stimulo-fugal*, or better, a *stimulo-concurrent character*.

Here I may call attention to the fact that both the neuroblasts and placode cells, as long as they have not developed dendrites, may shift with the nervous current, away from the excitation centre, contrary to that which occurs later with the ripe ganglion-cells.

It is clear, however, that the *functional connection* of the outgrowing axon cannot be determined by this process of irradiation alone, as was realized by Bok, who came to the conclusion that its final connection, its end-point, is determined by the chief law of neuro-biotaxis, namely, by a stimulative relationship between the place where the axon starts and the region in which it will end.

I shall return to this question of *selectivity* in the axonal endings at the end of my lecture. The stimulo-concurrent character of the axon and the stimulo-petal character of the dendrites and ripe ganglion-cells once being established, it puzzled me to find out the physico-chemical causes of these facts.

In the first place it may be asked how it is possible that in one and the same cell two opposed tropisms, a stimulo-petal tropism of the dendrites and a stimulo-fugal tropism of the axon, can occur?

Considering this question I felt inclined to believe that bio-electric influences, a species of galvano-tropism, exercised a leading influence here.

I thought so, *firstly* on account of the electric phenomena well known in nerve physiology, *secondly* on account of the opposite polarized character of dendrites and axons, and at last on account of

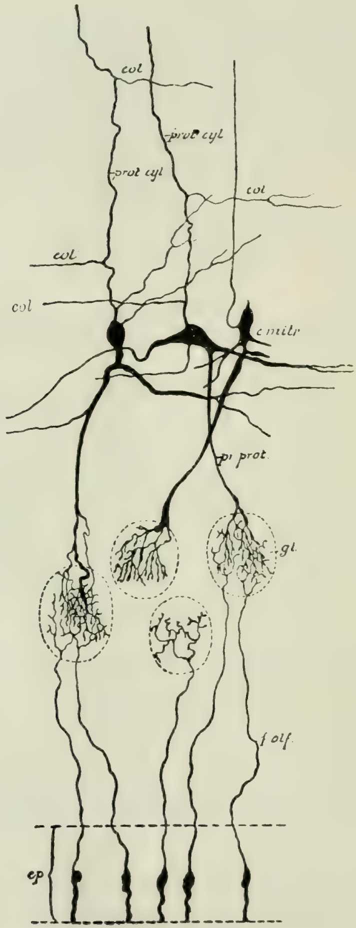

Fig. 4.—After Van Gehuchten. Notice the perpendicular course of the collaterals (col.) on the axis cylinders (prot. cyl.) of the mitral cells.

the fact that collaterals of axons very often run (at the beginning) exactly perpendicular to the mother axon, as for instance is seen in the drawings given by our too early deceased colleague, Van Gehuchten. Fig. 4 shows that collaterals grow perpendicularly from the axon, just as the axons of the young neuroblasts run perpendicular to the activating bundle.

A similar process, reminding us forcibly of bio-electric fields, is demonstrated by the perpendicular position of the dendrites of the Purkinje cells on the parallel fibres in the cerebellum (see fig. 5).

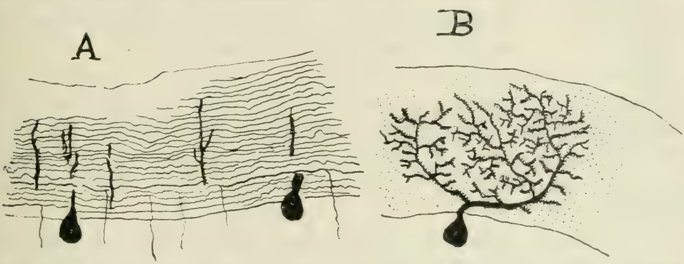

FIG. 5.—A, section through the molecular layer of the cerebellum of a cat, parallel to the parallel fibres of the layer. B, section perpendicular on the parallel fibres showing the spread of the dendrites of the Purkinje cells in one plane, perpendicular to the parallel fibres.

Analogous relations are found in the spinal cord of the lamprey where the dendrites of the motor cells stand perpendicular on the unmedullated longitudinal tracts (Tretjakoff).

The fact that many of these dendrites reach the periphery of the cord, where they may end in thickenings, has suggested to Tretjakoff the idea that their arrangement is due to assimilatory influences, since the spinal cord of the lamprey does not contain blood-vessels and the assimilatory substances come from the perimedullary vessels. Although I do not doubt that their close relation to the margin of the cord may have to be explained in this way, it does not seem probable to me that the *perpendicular position on the axis* of the cord must be attributed to this alone, since the contact with the periphery might also be obtained if the dendrites spread in different levels, most easily even in a fan-like way to the dorsal and ventral surface in a level parallel to the longitudinal axis of the cord, since the postero-ventral dimension of the cord is only very small in this animal. Besides the spreading of dendrites perpendicular to unmedullated fibres occurs also in the molecular layer of the cerebellum (*vide supra*), where such a spreading is not influenced by peripheral blood-supply since the molecular layer itself already contains a number of capillaries before the typical arrangement of the dendrites of Purkinje cells arises, which only takes place after birth (Addison).

It thus seemed to me that the neuro-biotactic process is based on bio-electric principles and that we have to do here with a sort of galvanotropic process (1908 and 1917).

Now by galvanotaxis or galvanotropism we understand the phenomenon that a living being, or part of it, when influenced by a constant electric current of a very low potential, has the inclination to turn to a certain pole, in most cases to the negative pole (the cathode).

Thus the roots of plants grow towards the negative pole, unicellular animals shift in the same direction, and the tentacles of a medusa move towards the negative pole (towards the cathode).

The interesting point, however, is that *this process is reversible.*

If the object, as for instance the roots of growing plants or unicellular animals, be brought into a stronger solution of potassium salts the tropism will be reversed and the object may turn towards the positive pole (the anode), instead of to the negative one.

This has probably to be explained by the influence of the electrolytes in the solution on the electrolytes in the vegetable skin, the presence of which also accounts for the blaze-currents, described by A. D. Waller.

The reversal of the tropistic process, then, must probably be explained by surface condensations of electrolytes, such as colloidal-chemistry shows us.

For we know from Hardy and others that albumin-like materials also show a shifting in a galvanic current, the process of *cataphoresis*, and it is very interesting that this cataphoresis may also be demonstrated experimentally with the nerve constituents, as has been shown first by Hermann and confirmed by Ingvar and myself. This shifting of colloidal materials can be reversed by acids, and directed towards the negative pole (cathode). Now there is a good deal in these reversible processes that seems to be applicable to the nervous system in connection with the physiology of excitation and conductivity.

In the first place, we know that a part of the surface of a nervous leader, in excitation, forms a cathode with regard to its surroundings which so to say represent an *anodic field* with regard to the surface of the excited centre.[1] (Fig. 6.)

The importance of this fact for the formations in the nervous system has been confirmed by Child, who also proves to be an adherent of the theory of bio-electric potentials as leading factors in the formation of the neuron (see p. 145 of this paper).

The nerve cells, that are now situated in the surroundings of that electro-negative excitation centre, first produce (probably on account of a change in surface tension, as is also supposed by Marinesco) a positive offshoot, corresponding to the irradiation of the nervous current from the excitation centre. This first kationic offshoot is the axon.

[1] Which inside is also positive.

This growing away from the excitation centre may be followed by a shifting of the cell itself in the same direction, so long as no dendrites occur, as for instance is seen with very young neuroblasts and placode cells.

Child has recently confirmed this point in my conclusions, emphasizing that ([17], p. 187) the region of origin of the axon is electrically determined in each cell by the electric currents of its environment.

It seems probable to me, that positive ions such as potassium act an important part in the axonal growth just as different concentrations of electrolytes act an important part in the nervous current itself, as has been demonstrated by Macdonald.

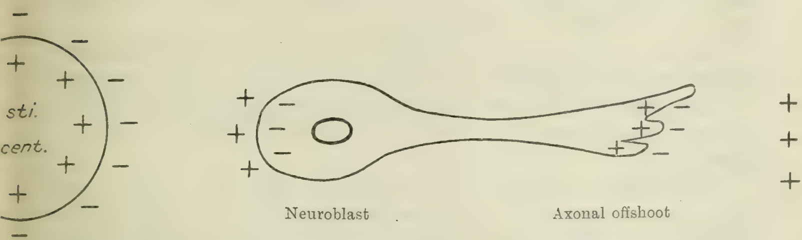

Fig. 6.—Eventual division of electrolytic potentials in the neuroblast (right) during the outgrowth of the axon by an action current.

We know that the axon more than other parts of the neuron contains large quantities of potassium compounds, as was pointed out by Macdonald, Macallum and Menten, independently of each other and in different ways. This high percentage of potassium must favour the stimulo-concurrent character of the nerve fibrillæ lying in it, that is the stimulo-concurrent growth of the axon. Consequently, just as Waller in his "Signs of Life" (p. 148) is inclined to consider the action current of the nerve as affected by kationic transport from the seat of action, so I consider even the outgrowth of that axon as being greatly influenced by such a kationic transport. *The outgrowth of the axon apparently is influenced by the action current*, which may also explain the influence of slight stimulations on regeneration.

And this is not so strange after all, since we know that the function of an organ and the growth of an organ are only two different aspects of the same thing.

Although I could not expect that this conception of the electric

character of the formation of the axon—which was a mere hypothesis until some months ago—should so soon be confirmed experimentally, I am in the happy position now of being able to state that the electric galvanotropic character of the outgrowth of the axon has been confirmed experimentally in embryonic tissue in serum cultures, in Harrison's laboratory, by our Swedish colleague, Sven Ingvar [44], who found that such an outgrowth may be determined by a constant galvanic current of a strength of 2 to 4 billionths of an ampère, density approximately $\frac{1}{1000}$ to $\frac{1}{2000}$ δ, non-polarizable electrodes.

So much as to the formation of the axon. At a much later stage the *dendrites* arise, and somewhat later still the cell body itself may commence to shift in an *opposite* direction to the axon, namely, towards the centre of excitation (thus stimulo-petal).

The stimulo-petal, cathodic tropism of the dendrites and of the perinuclear cell-plasm coincides with the appearance of the substances known as Nissl-bodies, which contain oxydases and catalases, and consequently only takes place when the neuron is in a far more advanced condition of development.

This tropism of the dendrites and the cell itself is in perfect harmony with the phenomena of excitation and contraction at the cathode according to Pflüger's law, as evinced in any protoplasm which, for instance, has been found for amœba by Loeb and Maxwell, and causes these parts to go to the field of excitation.

That the galvano-tropism of the dendrites is actually opposite to the galvano-tropism of the axon has also been confirmed experimentally by Ingvar in his serum cultures of the chick embryo.

If we consider the outgrowth of dendrites and the shifting of the ripe cell body as a tropism in the direction of the negative excitation surface, we must see whether there is any influence in the cell or dendrites that may make their tropism opposite to that of the axon in a galvanic field.

Now in trying to explain this opposite tropism of the dendrites (and of the ripe ganglion-cell), we are immediately struck by the fact that this cathodic shifting only occurs with those parts of the neurone which contain Nissl substance.

The axon, which contains no Nissl substance, exhibits a shifting in the opposite direction. That, indeed, this Nissl substance may have something to do with the cathodictropism of the dendrites and ripe cells, is further made probable by the fact that as long as the young

neuroblast contains no Nissl substance[1] it does not show the cathodic tropism, but even the opposite of it, and that the cathodic tropism occurs only when chromidial substance is developed in the cell which runs nearly parallel with the formation of the dendrites. It fails also to occur with placode cells, which contain no chromidial substance.

What then may be the influence of the chromidial substance on this process?

Now it seems that Hardy's work on cataphoresis, which, according to Greeley, may be applied to intra-protoplasmatic colloidal suspensions, furnishes us with the solution of this problem. We know that the Nissl substance during life (Cowdry), particularly during the development of the cells (van Biervliet), is in a more or less fluid condition, and that it surrounds the fibrillar substance.

This chromidial substance is a very complicated one, as has been demonstrated by Holmgren and Marinesco, of whom the latter working in Mott's laboratory showed the presence of *oxydases* in it. Thus much, however, is certain, that it is an acid derivative (a compound of nuclein acids with iron), and its acid character is also demonstrated by the fact that it can be coloured only by a basic strain.

According to Hardy's researches however, acids favour the shifting towards the cathode of the colloidal fibrillæ that are suspended in it.

Also the liberation and accumulation of the Nissl substance at the pole of the neurone that is opposite to the potassium accumulation agrees with electrolysis.

The late appearance of this chromidial substance in the cell-body, in which it appears only when the axon has already extended its growth over a considerable distance, would also make us understand the late formation of the dendrites. It thus seems that the developmental character of the whole neurone may be explained by bio-electric forces, and is in perfect harmony with the bio-electric phenomena known in nerve physiology: the growth of the axon depending on the action current, whereas the formation and contraction of dendrites is homologous to the cathodic outgrowth and contraction of pseudopodia, in a galvanic current and is favoured by metabolic processes, related to stimulation.[2]

[1] Whether the granulations which G. Levi saw ([46], p. 169) in his migrating cells, when they shift are to be compared with chromidial substance I do not know. I hope this excellent Italian histologist will continue his experiments which seem to me of utmost importance.

[2] That the stimulo-petal shifting of the dendrites and ripe cell-body is favoured by special chemical activities occurring in the dendrites and perikaryon is made probable by the fact

Mono-axonism and Polydendritism.

There are more characteristics of the nerve-cell that may be considered and explained from this standpoint. The *first* is that only one axon leaves the cell,[1] whereas a large number of dendrites may grow out from the cell-body in any direction towards several centres of stimulation (*mono-axonism and polydendritism*).

To explain the *mono-axonism* we must first realize that, with a polar tropism, as the galvano-tropism pre-eminently is, an object under the influence of a bio-electric current places itself so that the influence is equally great on both sides of the object. Then only does a condition of equilibrium occur. Thus the perpendicularly, stimulo-concurrent outgrowing axons of neuroblasts (fig. 3), as well as the perpendicular position of the collaterals upon the axis-cylinder (fig. 4), are natural consequences of the perfectly polar irradiation current. If now two (or more) different excitation centres simultaneously activate one cell, we may expect to meet with only one axis cylinder in the resultant line of both (or more) current directions (in the resultant of the bio-electric field), since it is only in this line that an equal influence on both sides of the growing axon is realized.

But what will happen, if two or more activating centres exercise their influence, *not simultaneously, but after each other upon the same cell-body?*

One of these activating centres will be necessarily the first and causes the formation of the axon hillock.

When, however, an axon hillock has commenced to grow, *i.e.*, when an efferent zone of fibrils has been formed in the cell-body, we may expect that the opportunity, offered by this zone, on account of

that their constituents act a much greater part in the assimilation of oxygen (on account of the oxydases and catalases occurring in the Nissl substances) and not in the axon.

Stimulation and oxygenation probably run parallel here (stimulation setting free the catalases) and sustain not only each other (Lillie) but also the neurobiotactic process as is suggested to me by Sir Fred. Mott. The influence of metabolism on the growth of dendrites cannot be denied and is proved by the Cajal cells and the perimedullary dendrites in the lamprey (see above) which has no intraspinal blood-vessels and where the dendrites spread very richly on the periphery of the cord where they end in thickenings, just as ependymal and glia cells do, as if to have more ample contact with assimilatory substances. Since stimulation and assimilation run frequently parallel this may also act a part in the direction of the dendrites to the source of stimuli. That metabolic processes may influence the galvano-tropism has also been stated in lower organisms, since Terry found that "the galvano-tropism of volvox depends upon its state of chlorophyll metabolism" ([77], p. 243).

[1] The horizontal cells of Cajal may exhibit two or more axis cylinders. These do not proceed however from the cell-body, but originate at great distances from each other, from different dendrites.

its greater conductivity, is so favourable, that every new current that runs over the cell, instead of forming a new axon in another place of this cell, will make use of the opportunity of better conductivity, already present, the more so since on account of the originally irradiating character of nervous currents, these currents, if they reach the cell, will necessarily always cover the whole of it, and thus necessarily meet the axon hillock. The first formed zone of efferent fibrils, the axon-hillock,[1] becomes thereby the favourite conveying path for all the stimuli that run over or through the cell-body.

The relations for the formation of dendrites are, however, altogether different.

When a stimulus appears in the vicinity of the cell, the most adjacent part of the protoplasm of such a cell may shift in the direction of that stimulus. With the equality of that protoplasma and the ubiquity of the Nissl substance in the whole cell (with the exception of the axon hillock) this process is, of course, not restricted to one field, and finds no place of predilection, except the one that is nearest by. Centres of excitation situated elsewhere may, and even must, cause new protoplasmatic outgrowth in their own direction, if the dendrites that have previously grown out are not on their way.

So we find that mono-axonism is a result of the polar localization of the anodotropic part of the cell, whereas polydendritism is based on the fact that the cell-plasm has no place of predilection and is everywhere sensitive to the cathodic tropism.

The Selectivity in the Formation of Neuronal Connections.

I have already said that the anatomical relations as found in the nervous system and the processes of cell shifting clearly point to the fact that the relationship which determines the connections is the synchronic or immediate successive function of the elements.

This fundamental law of neurobiotaxis does not merely show us that the well-known law of association in psychology is also a neurobiotactic law, but it also shows how wonderfully polar the whole character of the tract formation is, and how well it fits in with the class of bio-electric phenomena. For the purpose of explaining this selectivity I must draw attention to the following points.

[1] The polar character of which has been demonstrated by Held to coincide with the diplosome-centre.

We may assume that a state of excitation, once started in a budding axon, proceeds rapidly and a strong current of internally positive potential reaches the buddingcone of the axon. That this is more than a mere supposition follows from the researches of Scaffidi[1] (and Viale) who proved that, if a nerve after section starts to regenerate its budding cone shows a positive potential current, that reminds us of an action current and may be called regeneration current or—as I do in embryonic tissue—a *growth current.*

If we now assume that in the neighbourhood of this budding cone several nerve-cells are situated, one of which is already in a state of electric dissociation, while the others are not, it will be evident that the cell which is in a state of ionization is the only one that presents a selective point to the budding axon.

The potential, that continues along the budding axon, may find its natural counterpart only in the already ionized cell, and not in cells which are not stimulated and which are consequently indifferent objects, not presenting any predilection.

The principal part in establishing the connections between the growing axon and the cell or dendrites with which it is going to be connected may be played by the raised *positive surface potential* which appears near the cell shortly after some previous stimulation and which immediately follows its stimulation (see fig. 6). Or, in other words, the growth-current of the budding axon will find a place of predilection in a neurone that has been in action shortly before, just as with ships in a battle line one ship sails in the wake of the ship that precedes.

In the physiology of nervous currents there are several facts which are in favour of the supposition that an immediately prior excitation exercises a directing influence upon the course of new excitations occurring in the nervous system, since Sherrington emphasizes, "that the threshold of a reflex is lowered by the excitation just preceding its own," and this also explains why a connection just established between two neurones (a synapse) "is an apparatus for co-ordination and introduces *a common path*," i.e., of several other excitations ([75], pp. 184, 351).

This principle of selectivity based on the influence of a just preceding state of function as forming a centre of attraction for other

[1] *Zeitsch. f. allg. Physiologie*, 1910, Bd. xi, S. 345 : "Die Schnittflächen der Nerven, welche nach der Durchtrennung *in situ* bleiben, sowohl nach wenigen Minuten als nach verschiedenen Monaten fast immer positiv u.s.w. Die positive Ladung der Schnittflächen verschwindet rascher nach der einfachen Durchtrennung, d. h. wann Verschmelzung folgt."

nervous currents or budding axones, becomes of use in a most interesting way, in making us understand the connections of the nervous system with the muscles. Bok (1917) has shown that the connection between certain muscles with sometimes widely distant places of the central nervous system, has to be explained by the fact that a contraction of a muscle (the formation of which precedes the formation of nerve roots) acts tropistically upon the central nerve fibres.

A contraction, or an analogous condition, raised by some external non-nervous circumstance, attracts action currents which happen to be present at that moment in the longitudinal tracts of the central nervous system, thus leading at the same time their growth.

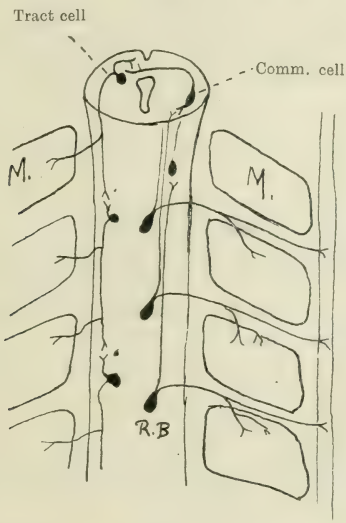

FIG. 7.—Relation of the spinal cord to the myotomes (M.) and skin. On the right hand, R.B., Rohon-Beard's sensory cells for skin and muscle sensibility. To the left are collaterals forming motor nerve-roots. (After Herrick and Coghill.)

In accordance with this conception is the fact that the first motor roots as they occur in invertebrates and as Coghill found them in larvæ of amphibia, originate as collaterals of longitudinal tracts (fig. 7).

Only later the neuroblasts lying next to such longitudinal tracts are activated to form axons, that replace these collaterals and form the

real motor roots. Hence it follows that originally the myotomes attract the action currents present in the central nervous system immediately after the moment of their contraction, or, in other words, *that the process of contraction is originally only integrated, not caused by those central currents.*

Later on, when the connection of the roots with the myotome has been established, the contraction may of course be caused by those central reflexes. Now I said that cat-ions as potassium probably exercise a considerable influence in the growth of the axon just as according to Macdonald and Waller it does in the action current of the nerve. Similarly we find that potassium may act a part in the process of the entering of nerve-fibres into the muscle, since Boeke found the nerve-fibrils to end in the anisotropic layer of the muscle in which, according to Macallum's reactions, potassium is localized (see also Woerdeman). I hardly need to say that in this respect also the researches of Howell, and specially those of Zwaardemaker, concerning the rôle of potassium in the transmission of stimulation are of utmost importance.

It is probable that in embryonic objects the proliferation of muscle tissue has the same influence as the function of adult tissue and that proliferating muscle-tissue thus may attract irradiations of the nervous currents from the spinal cord, in the same way as the proliferating connective tissue attracted the collaterals of the roots and even of the lateral funiculi in the experiments of Cajal.

Child recently has called our attention to the probability that during the evolution of tissue electric conditions occur very similar to those occurring in stimulation.

The sequence however in which this embryonic proliferation takes place must be caused by engrammatic conditions the character of which as yet entirely escapes our conception.

It is evident that there are many more questions in nerve histology and nerve physiology that may be considered from this standpoint, which, I hope, may be useful in giving the missing link between the structural and functional features of the nervous system.

I am afraid, however, that I have taken already too much of your attention and I will finish here by stating—what perhaps you have already stated yourself, when listening to me—that a large part of the researches forming the stones that enabled me to build this theory are given by others, and not least by your own countrymen.

POSTSCRIPT.

It was only after delivering this lecture that I came to study Child's valuable book, " Origin and Development of the Nervous System, from a physiological viewpoint," a very interesting contribution to our knowledge of development of pattern.

I have not been able to discuss its contents fully in this paper, though I have added some references to it. I will not omit recommending it to those who are interested in the directing influence of electric potentials, and I am glad that Child and I fully agree in the importance of these potentials.

Reserving a fuller discussion for a later paper, I will only add that I do not agree with him in ascribing the development of ventral roots and dorsal roots to two principally different types of polarization—his first and second type.[1] Neither do I agree with his supposition "that it is not necessary to assume that the dendrites react differently to the electric current than the axon." ([17], p. 195.)

Apart from my own deductions on this point I may refer to Ingvar's statement that the offshoots that grow to the cathode and those which grow to the anode in his experiments show a difference of character ([44], p. 198), and further to a personal communication of this author to me that it seemed to him that one of these offshoots was the axon, and the opposite one a dendrite.

I further must state that the division of electrolytes which Child gives in his fig. 57, as presenting my idea about this matter, does not represent my idea.

I have in the paper quoted by Child purposely not given a schematic indication of the division of electrolytes in the cell, because I could not do so, and certainly would not like to express it in such a simple scheme. I would rather give it (but still with great hesitation) as it is given in fig. 6 of this paper, for the development of the axon.

It seems to me that the last-mentioned scheme agrees with the accumulation of the more or less acid chromidial substance inside the pole of the neurone opposite to the axon-formation, where later the dendrites occur, and where oxidation, and also acid constituents (that is, negative potentials) are really found later on, as is to be expected from what we know of electrolytic dissociations. I am afraid, however,

[1] Such different types, apart from the artificial assumption of their existence in the nervous system, are, even if they were possible, doubtful, as long as the question of plasmodesmic pre-formed paths (that is of tissue continuity) is not sufficiently settled. (Compare Paton and Neal.)

that at present we are not yet able to state with any amount of certainty the exact division of potentials in the tissue even in serum culture experiments, since the applied potentials may be reversed by membranes or Helmholtz-Quincke layers, to which Child also calls attention.

The chief point is that we agree that electric potentials are the moving factors. As far as concerns their details the neurobiotactic phenomena will offer still many difficulties and controversies, the whole problem being a very difficult one.

A point in which we are especially grateful to Child is his pointing out in connection with Lillie's work the relation between oxidative processes and growth in certain directions (gradients). Specially the relations of the dendrites as found in Petromyzon (Ammocœtes) comes thereby nearer to our conception. (See p. 135 and footnote [2] on pp. 139 and 140.

REFERENCES.

[1] Addison. "The Development of the Purkinje Cells and of the Cortical Layers in the Cerebellum of the Albino Rat," *Journ. Compt. Neurol.*, 1911, **21**, 459.

[2] Alcock and Lynch. "On the Relation between the Physical, Chemical and Electrical Properties of the Nerves. Part iv: Potassium, Chlorine and Potassium Chloride," *Journ. of Physiol.*, 1911, **42**, 107.

[3] Ariëns Kappers. "Die phylogenetische Verlagerungen der motorischen Oblongata-Kerne, ihre Ursache und ihre Bedeutung," *Neurolog, Centralbl.*, 1907, and *Rapport du Congrès Internat. de Psychiat. et Neurol.*, Amsterdam, 1907.

[4] *Idem.* "Weitere Mitteilungen bezüglich phylogenetischen Verlagerungen der motorischen Hirnnervenkerne. Der Bau des autonomen Systemes," *Folia Neurobiol.*, 1908, **1**, 157.

[5] *Idem.* "Weitere Mitteilungen über Neurobiotaxis. Die Selectivität der Zellenwanderung. Die Bedeutung synchronischer Reizverwandtschaft, &c.," *Folia Neurobiol.*, 1908, **1**, 507.

[6] *Idem.* "Ueber die Bildung von Faserverbindungen auf Grund von simultanen und sukzessiven Reizen," *Bericht des* 111 *Congr. f. exper. Psychiol.* in Frankfurt-am-Main, 1908.

[7] *Idem.* "The Structure of the Autonomic Nervous System compared with its Functional Activity," *Journ. of Physiol.*, 1908, **37**, 139.

[8] *Idem.* "Phenomena of Neurobiotaxis in the Central Nervous System," Section of Anatomy and Embryology, *XVIIth International Congress of Medicine*, London, 1913, Sect. 1, pt. ii, 109.

[9] *Idem.* "Further Contributions on Neurobiotaxis. IX: An Attempt to Compare the Phenomena of Neurobiotaxis with other Phenomena of Taxis and Tropism. The Dynamic Polarisation of the Neurone," *Journ. of Comp. Neurol.*, 1916, **17**, 27, 261.

[10] Balfour. "On the Development of the Spinal Nerves in Elasmobranch Fishes," *Proc. Roy. Soc. Lond.*, 1876.

[11] Bancroft. *Journ. Exper. Zool.*, 1904, **1**, 289.

[12] van Biervliet. "La substance chromophile pendant le cours de développement de la cellule nerveuse," *Le Névraxe*, 1900, **1**, 33.

[13] Boeke. "On the Structure of the Light-percepting Cells in the Spinal Cord, the Neurofibrillæ in the Ganglion Cells and the Innervation of the Striped Muscles in *Amphioxus lanceolatus*," *Proc. Kon. Akad. van Wetensch.*, Amsterdam, 1902, **5**, 350.

[14] Bok. "Die Entwicklung der Hirnnerven und ihrer zentralen Bahnen. Die stimulogene Fibrilation," *Folia Neurobiol.*, 1915, **9**, 475.
[15] *Idem.* "Stimulogeneous Fibrillation. The Cause of the Structure in the Nervous System," *Psych. en Neurolog. Bladen*, Amsterdam, 1915, **19**, 393.
[16] *Idem.* "The Development of Reflexes and Reflex Tracts. I: The Reflex Circle," *Psychiat. en Neurol. Bladen*, Amsterdam, 1917, **21**, 281.
[17] Child. "The Origin and Development of the Nervous System from a Physiological View-Point," Chicago, 1920.
[18] Coehn and Barratt. "Ueber Galvanotaxis vom Standpunkt der Physikalischen Chemie," *Zeitschr. f. allgem. Physiol.*, 1905, **5**, 1.
[19] Coghill. "The Primary Ventral Roots and Somatic Motor Column of Amblystoma," *Journ. of Comp. Neurol.*, 1913, **21**, 121.
[20] *Idem.* "Correlated Anatomical and Physiological Studies of the Growth of the Nervous System of Amphibia," *Journ. of Comp. Neurol.*, 1914, **24**, 161.
[21] Cowdry. "The Development of the Cytoplasmic Constituents of the Nerve Cells in the Chick. Mitochondria and Neurofibrils," *Amer. Journ. Anat.*, 1913-14, **14**, **15**, 389.
[22] *Idem.* "The Relations of Mitochondria and other Cytoplasmic Constituents in Spinal Ganglion Cells of the Pigeon," *Internat. Monatschr. f. Anat. u. Physiol.*, 1913, **29**, 473.
[23] *Idem.* "The General Functional Significance of Mitochondria," *Amer. Journ. of Anat.*, 1916, **19**, 423.
[24] Dart. "A Contribution to the Morphology of the Corpus Striatum," *Journ. of Anat.*, 1920, **55**, 20.
[25] Davidson Black. "The Motor Nuclei of the Cerebral Nerves. A Study of the Phenomena of Neurobiotaxis. Part I: Cyclostomi and Piseer," *Journ. of Comp. Neurol.*, 1917, **27**, 467. "Part II: Amphibia," *ibid.*, 1918, **28**, 379. "Part III: Reptilia," *ibid.*, 1920, **32**, 61.
[26] Dustin. "Le rôle des tropismes et de l'odogenèse dans la régénération du système nerveux," *Arch. de Biol.*, 1910, **25**, 269.
[27] Gassner. "Der galvano-tropismus der Wurzeln," *Botanische Zeitung*, 1906, pts. ix-xi.
[28] Grandry. "Recherches sur la structure intime du cylindre de l'axe et des cellules nerveuses," *Bull. de l'Acad. Roy. des Sciences de Belgique*, 1868, 2 ser., **25**, 304.
[29] *Idem.* *Journ. de l'Anat. et de la Physiol.*, 1869, **6**, 289.
[30] Greeley. "Experiments on the Physical Structure of Protoplasm of Paramecium and its Relation to the Reactions of the Organism to Thermal, Chemical and Electrical Stimuli," *Biol. Bulletin*, Woods Hole, 1904, **7**, 3.
[31] Hamburger. "Osmotischer Druck und Ionenlehre," Wiesbaden, 1904, **3**, 68.
[32] Hardy. "On the Coagulation of Proteid by Electricity," *Journ. of Physiol.*, 1899, **24**, 288; *Proc. Roy. Soc. Lond.*, 1900, **66**, 110.
[33] Harrison. "The Outgrowth of the Nerve Fibre as a Mode of Protoplasmic Movement," *Journ. Exper. Zool.*, 1910, **9**, 787.
[34] Held. "Die Entwicklung des Nervengewebes bei den Wirbeltieren," Leipzig, 1909.
[35] Hermann. "Eine physikalische Erscheinung am Nerven," *Pflüger's Archiv*, 1897, **67**, 240.
[36] Herrick. "The Morphology of the Forebrain in Amphibia and Reptilia," *Journ. of Comp. Neurol.*, 1910, **20**, 486.
[37] Herrick and Coghill. "The Development of Reflex Mechanisms in Amblystoma," *Journ. of Comp. Neurol.*, 1915, **25**, 65.
[38] van Herwerden. "Ueber die Nuclease-Wirkung auf tierische Zellen. Ein Beitrag zur Chromidienfrage," *Arch. f. Zellforschung*, 1913, **10**, 431.
[39] His. "Die Entwicklung der ersten Nervenbahnen beim menschlichen Embryo," *Arch. f. Anat. u. Physiol.*, Anat. Abth., 1887, 369. (See also "Die Entwickelung des menschlichen Gehirnes," Leipzig, 1904.)
[40] His, jun. "Die Entwickelung des Herznervensystems bei den Wirbeltieren." *Abhandlungen der Kön. Sächs, Akad. der Wissenschaften*, Math.-Phys. Classe **23**, 1893.

[41] v. d. Horst. "Die motorischen Kerne und Bahnen in dem Gehirn der Fische; ihr taxonomischer Wert und ihre neurobiotactische Bedeutung," *Tijdschrift der Nederl. Dierkund. Vereenigingl* (2), 1918, **16**, 169.

[42] Howell. "Vagus Inhibition of the Heart in its Relation to the Inorganic Salts of the Blood," *Amer. Journ. of Physiol.*, 1905-06, **15**, 280.

[43] Howell and Duke. "The Effect of Vagus Inhibition on the Output of Potassium from the Heart," *ibid.*, 1908, **21**, 51.

[44] Ingvar. "Reaction of Cells to the Galvanic Current in Tissue Cultures," *Proc. Soc. Exper. Biol. and Med.*, 1919-20, **17**, 198.

[45] Keller. "Die Elektrizität in der Zelle," Wien, 1918.

[46] Levi, G. "Connessioni e struttura degli elementi nervosi fuori dell'organismo," *Reale Accad. des Lincei*, ser. v, 1917, **12**, 142.

[47] Lillie. "The Relation of Stimulation and Conduction in Irritable Tissues to Changes in the Permeability of the Limiting Membranes," *Amer. Journ. Physiol.*, 1911, **28**, 197.

[48] *Idem.* "The Formation of Structures resembling Organic Growths by Means of Electrolytic Local Action in Metals and the Physiological Significance and Control of this Type of Action," *Biolog. Bull.*, Woods Hole, 1917, **33**, 135.

[49] Loeb. "Zur Theorie des Galvano-tropismus. III: Ueber die polare Erregung der Hautdrüsen von Amblystoma durch den constanten Strom," *Pflüger's Archiv*, 1897, **65**, 308.

[50] *Idem.* "Vorlesungen über die Dynamik der Lebenserscheinungen," Leipzig, 1906, 171.

[51] Loeb und Budgett. "Zur Theorie des Galvano-tropismus. IV: Mitteilung über die Ausscheidung electropositiver Ionen an der äusseren Anodenfläche protoplasmatischer Gebilde als Ursache der Abweichungen vom Pflügerschen Erregungsgesetz," *Pflüger's Archiv*, 1897, **65**, 518.

[52] Loeb und Maxwell. "Zur Theorie des Galvano-tropismus," *Pflüger's Archiv*, 1896, **63**, 121. (See also Loeb und Gerry, "Zur Theorie des Galvano-tropismus. II: Versuche an Wirbeltieren," *Pflüger's Archiv*, 1897, **65**, 41.)

[53] Macallum. "On the Distribution of Potassium in Animal and Vegetable Cells," *Journ. Physiol.*, 1905, **32**, 95.

[54] *Idem.* "Die Methoden und Ergebnisse der Mikrochemie in der biologischen Forschung," *Ergebnisse der Physiologie*, 1908, **7**, 552.

[55] *Idem.* "Surface Tension and Vital Phenomena," University of Toronto Studies, No. 8, Physiological Series, 1912.

[56] Macallum and Menten. "Distribution of Chlorides in Nerve-cells and Fibres," *Proc. Roy. Soc. Lond.*, 1906, **77B**, 165.

[57] MacDonald. "The Injury Current of Nerves: The Key to its Physical Structure." Report of Thomson-Yates Laboratory, 1902, **4**, 213.

[58] *Idem.* "The Structure and Function of Nerve-fibres," *Proc. Roy. Soc. Lond.*, 1905, **76B**, 322.

[59] Marinesco. "Plasticité et amiboidisme des cellules des ganglions sensitifs," *Revue neurologique*, 1907, **15**, 1109.

[60] *Idem.* "Recherches anatomo-cliniques sur les névromes d'amputation douloureux," *Phil. Trans. Roy. Soc. Lond.*, 1919, **209B**, 229.

[61] *Idem.* "Recherches histologiques sur oxydoses and études histologiques sur les oxydases et les peroxydases," *Compt. rend. de la Soc. de Biol.*, 1919, **82**, 98 and 258.

[62] Mott. "The Degeneration of the Neurone." Croonian Lecture, Roy. Coll. Phys., 1900.

[63] *Idem.* "Studies in the Pathology of Dementia Præcox," *Archives of Neurol.*, 1921, **8**, 1.

[64] Müller-Hettlingen. Ueber galvanische Erscheinungen an keimenden Samen," *Pflüger's Archiv*, 1883, **31**, 193.

[65] Neal. "Nerve and Plasmodesma," *Journ. of Comp. Neurol.*, 1921, **33**, 65.

[66] Paton. "The Reactions of the Vertebrate Embryo to Stimulation and the Associated Changes in the Nervous System," *Mitt. a. d. Zool. Station zu Neapel*, 1906-08, **18**, 535.

[67] RAMON Y CAJAL. "La rétine des vertébrés," *La Cellule*, 1892, **9**, 119.
[68] *Idem.* "Die Regeneration der Nervenfasern" (originally in Spanish), Trabajos, &c., **4**.
[69] *Idem.* "Textura del sistema nervioso del hombre y de los vertebrados," 1899, **1**, 560.
[70] *Idem.* "Algunas observacionas favorables a la teoria neurotropica," Trabajos, 1908, **7**, 63.
[71] ROTHERT. "Die neuen Untersuchungen Über den Galvano-tropismus der Pflanzenwurzeln," *Zeit. für allg. Physiol.*, 1907, **7**, 142.
[72] SCHELLENBERG. "Untersuchungen Über den Einfluss der Salze auf die Wachstumsrichtung der Wurzeln, zunächst an der Erbsenwurzel," *Flora*, 1906, **96**, 474.
[73] SCOTT. "On the Structure, Microchemistry and Development of Nerve-cells with special reference to Nuclein Compounds," *Trans. Canadian Institute*, 1899, **6**, 405.
[74] SEDGWICK. "On the Inadequacy of the Cellular Theory of Development, and on the Early Development of Nerves, particularly of the Third Nerve, and of the Sympathetic in Elasmobranchii," *Quart. Journ. of Microscopical Science*, 1895, **37**, 87.
[75] SHERRINGTON. "The Integrative Action of the Nervous System," London, 1906.
[76] SMITH (ELLIOT). "A preliminary note on the Morphology of the Corpus Striatum and the origin of the Neopallium," *Journ. of Anat.*, July, 1919, **53**, pt. iv.
[77] TERRY. "Galvanotropism of Volvox," *Amer. Journ. Physiol.*, 1905-6, **15**, 235.
[78] TRETJAKOFF. "Das Nervensystem von Ammocoetes; I: Das Rückenmark," *Archiv f. mikr. Anat.*, 1909, **73**, 607, pl. 24, fig. 11.
[79] *Idem.* "Das Nervensystem von Ammocoetes; II: Das Gehirn," *ibid.*, 1909, **74**, 636.
[80] VERWORN. "Die polare Erregung der Protisten durch den galvanischen Strom," *Pflüger's Archiv*, 1889, **45**, 1.
[81] *Idem.* "Die polare Erregung der Protisten durch den galvanischen Strom (Fortsetzung)," *ibid.*, 1890, **46**, 267.
[82] *Idem.* "Untersuchungen Über die polare Erregung der lebendigen Substanz durch den constanten Strom; III Mitteilung," *ibid.*, 1896, **62**, 415.
[83] *Idem.* "Die polare Erregung der lebendigen Substanz durch den constanten Strom; IV Mitteilung," *ibid.*, 1897, **65**, 47.
[84] WOERDEMAN. "An Inquiry into the Distribution of Potassium Compounds in the Electric-organ of the Thornback (*Raja clavata*)," *Proc. Kon. Akad. van Wetensch.*, 1921, **23**, 842.
[85] ZWAARDEMAKER. "Die Bedeutung des Kaliums im Organismus," *Pflüger's Archiv*, 1919, **173**, 28.

A CLINICAL AND EXPERIMENTAL CONTRIBUTION TO THE PATHOGENESIS OF DISSEMINATED SCLEROSIS.

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THE investigations with which this paper deals were begun in the autumn of 1913, and resumed in 1920 after an interruption of five years caused by the War. At the time when our work was begun no evidence from the experimental side had been published which served to throw any light on the obscurity in which the pathogenesis of disseminated sclerosis has so long been shrouded. In October, 1913, a paper appeared by Bullock [2], who claimed to have transmitted the disease from man to rabbits, while in 1914 an exhaustive work was published by Siemerling and Raecke dealing with the morbid anatomy and histology of the disease. During and since the War other evidence was forthcoming in the direction of a solution of the problem confronting us, and it is mainly on this account that we have decided to publish the results of our investigations. It can at once be stated that our experimental results are almost entirely negative, and we are well aware that their publication may appear to some unnecessary. We feel, however, that negative evidence cannot be entirely disregarded, especially in view of the fact that much of the positive evidence which has recently appeared is to our mind unsatisfactory and unconvincing.

The clinical material on which this work was carried out was obtained from the wards either of the National Hospital, Queen Square, or of St. Thomas's Hospital. To the members of the staffs of both hospitals who gave us every facility for our investigations we wish to tender our grateful thanks.

Thirty-five cases have been investigated clinically and bacteriologically, while animal inoculations have been carried out in fifteen instances, in two cases with material obtained post mortem.

Clinical Study.

Although it is not our intention to enter into a detailed discussion regarding the differential diagnosis of disseminated sclerosis, nevertheless this aspect of the matter must not lightly be passed over. In more than one recent publication, for example, dealing with the transmissibility of the disease to animals, too little importance has been attached in our opinion to the clinical side. We are merely told that the case providing the material to be injected was one of "typical multiple sclerosis"; or else a brief clinical résumé is appended from which it is not always clear that the patient actually had this disease. In three cases investigated by us the provisional diagnosis of disseminated sclerosis was proved to be incorrect, and in two others (Cases IV and XIII) it remained doubtful. (*Vide* Appendix.) Of the three patients wrongly diagnosed, none of whom are included in our series, one proved to be a case of syringomyelia, while the other two were cases of cerebrospinal syphilis.

In no disease is accurate history-taking more important than in the one under consideration, and in a large majority of cases a diagnosis can be made from the history alone. The patient's description of the onset and character of the various symptoms is usually sufficient to make it clear that widely separated areas of the central nervous system have been affected at the same or at different times. This feature differentiates disseminated sclerosis from all other common diseases of the central nervous system with the *important exception of cerebrospinal syphilis*. Further, the history demonstrates that the course of the disease is not always slowly progressive and uneventful as was at one time supposed, but is more often interrupted by acute and unexpected disturbances. In more than half (54·3 per cent.) of our cases the onset was comparatively sudden, while in more than two-thirds (68·6 per cent.) some of the symptoms, but not necessarily the initial disturbance, came on acutely. In 85·7 per cent. of our cases the disease had run a discontinuous course, the symptoms pointing now to this and now to that locality of the brain or cord, appearing in an irregular and haphazard manner, and at greatly varying intervals of time. Remissions or quiescent periods, often of many years' duration, alternate with periods marked by the more or less rapid development of fresh disturbances in a manner which is bewildering in so far as it defies accurate anticipation.

We have taken care to select for the purposes of this investigation only those cases which presented evidences of *recent or progressive*

lesions. This selection excludes from the present review two types of case, first the non-progressive case, i.e, patients in whom the disease is in a state of arrest, the condition having remained completely stationary for several months or years, and secondly the so-called "cures," i.e., those in whom no trace remains of a previous pathological condition, as judged by an entire absence of both symptoms and physical signs. We wish to emphasize the fact that such cases occur not infrequently, and that patients, especially those in whom the initial disturbance was relatively sudden, may remain in perfect health for months or years at a time (*vide* Case XIX). How long this immunity can last we are not in a position to say, but in view of the fact that the existence of the non-progressive group is generally admitted, together with the circumstance that the initial stages of the disease are frequently overlooked or wrongly diagnosed, it seems possible at any rate that more cases than is commonly supposed recover from their early lesions and remain well permanently.

Although we desire to lay particular stress on the acute features of the disease, it must not be forgotten that a small number of cases begin insidiously and progress insidiously, while many cases also, which in the early stages were characterized by exacerbations and remissions, eventually assume a more chronic character and progress slowly downhill without new incidents. No hypothesis of the pathogenesis of disseminated sclerosis is satisfactory which fails to take into account the different clinical forms which the disease assumes in different individuals, or in the same individual at different stages of its development.

Clinically, therefore, disseminated sclerosis reveals itself in a haphazard series of relatively acute disturbances occasioned by focal lesions distributed at random both in the brain and cord, appearing at irregular intervals, and showing in their early stages a general tendency to improvement.

Sites of election have long been recognized in this disease, and it is well known that the white matter of the cord, as opposed to that of the brain-stem, is much more commonly affected than the grey. In consequence of this tendency to localize itself in certain areas, the disease presents clinical features which are said to be "typical." Nevertheless no single symptom or sign (e.g., "scanning speech") is of itself characteristic of the condition, but rather a combination of such symptoms and signs. We would further point out that a condition so kaleidoscopic in its symptomatology and clinical course as is

disseminated sclerosis seems peculiarly ill adapted for classification into types and sub-types so dear to the heart of many continental writers.

In the accompanying table is set forth the relative frequency with which certain symptoms and signs were found in our thirty-five cases. Besides indicating the multiplicity of the symptomatology of the disease, it shows that at any rate in the earlier stages the clinical picture departs widely from most text-book descriptions.

NUMBER OF CASES EXAMINED: THIRTY-FIVE.

	Number of cases	Percentage of total
Discontinuous course	30	85·7
Chronic progressive course	5	14·3
Paræsthesiæ	29	82·6
Loss or impairment of deep sensation	23	65·7
Loss or impairment of vibration in lower limbs (23 cases examined)	14	60·8
Disturbance of cutaneous sensibility	11	31·4
"Useless arm"	14	40·0
Extensor plantar response	32	91·4
,, bilateral	27	77·1
,, unilateral	5	14·3
Absent abdominal reflexes (bilateral)	27	77·1
,, ,, ,, (unilateral)	1	2·9
Spastic paraplegia	16	45·7
Sphincter disturbance	25	71·4
Nystagmus	26	74·3
Intention tremor of arms	15	42·6
Cerebellar inco-ordination of arms	15	42·6
Cerebellar gait	18	51·4
Tremor of unsupported head	13	37·1
Ataxic dysarthria	10	28·6
Pallor of optic disks (33 cases examined)	19	57·6
,, ,, ,, (bilateral)	15	45·5
,, ,, ,, (unilateral)	4	12·1
Sudden amaurosis	7	20·0
Severe bilateral visual failure	3	8·6
Diplopia	12	34·3
Rotatory vertigo	18	51·4
Defective emotional control	18	51·4

The most common symptom was paræsthesia, present in 32·6 per cent. of the cases. Although often transient, these subjective disturbances are absolutely definite and constitute one of the most characteristic features of the disease. They may be the first evidence of the pathological process, and although varying in their character and locality are rarely absent at one or other period of the illness. They are variously described as "tightness," tingling, numbness, boring pains (never lancinating pains as in tabes), aching, "rheumatism," "like water trickling down the skin," "like something tickling," coldness, "as if my legs were being scorched in front of a fire," pins and needles, "as if my body and legs were in plaster," &c. These

sensations were referred to the trunk and (or) limbs, and in two cases to the tongue, but were never associated with visceral disturbances such as occur in tabes. Except that they are far less frequently restricted to the peripheral segments of the limbs, they are comparable in every particular to the paræsthesiæ which form so notable a feature in the symptomatology of toxic degeneration of the posterior columns of the cord associated with pernicious anæmia (subacute combined degeneration), and they are to be regarded as evidence of morbid changes in the posterior columns.

Evidence of disturbance of deep sensibility in one or more limbs was obtained in two-thirds (65·7 per cent.) of our cases. Unfortunately vibration was not tested in every case, otherwise the proportion would probably have been greater. In the twenty-three cases in which vibration was tested, in fourteen (60·8 per cent.) loss or impairment of this quality of sensation was demonstrated in one or both of the lower extremities. We are, therefore, unable to agree with the statement current in most text-books that objective disturbances of sensation are rare in this disease. On the contrary we regard disturbance of deep sensation (i.e., compasses, tuning-fork, stereognosis, passive position and movement) as one of the most characteristic signs of the disease, and as evidence of involvement of the posterior columns of the spinal cord or of their upward continuations.

With regard to disturbance of cutaneous sensibility we agree with other authors that it is relatively uncommon; in our series it was present in less than one-third of the cases (31·4 per cent.).

Before leaving the subject of sensory disturbances we should like to draw attention to a symptom-complex, first described by Oppenheim, which may be called the "useless arm." The condition usually develops rapidly in the course of a few hours or days, with or without preliminary warnings in the shape of paræsthesiæ. As a rule it is unilateral, and the expression used by the patient to describe the condition is almost invariably the same, viz., "the arm is useless." Objectively there is profound loss of deep sensation in one of the upper extremities, e.g., no threshold to compasses, complete astereognosis, no response to vibration, and wild sensory ataxia with abolition of the sense of passive position and movement sometimes complete even at the shoulder-joint. Hence the patient says: "I lose my arm in bed," or "The arm has an existence apart from me." The limb is not paralysed, and provided the patient gazes fixedly at the arm when each muscle group is tested there is no loss of power, but

with closed eyes there is an apparent paresis, which is however entirely due to the falling out of afferent impulses from muscles and joints and a consequent inability properly to apply muscular power.

This curious condition may be the first and only evidence of disseminated sclerosis; in the present series it was not infrequently associated with nystagmus, more rarely with a transient impairment of tactile sensibility in the affected limb, and never with bulbar symptoms. The condition of the arm improved in every case, and cleared up entirely in Cases XIX and XXXIV. We regard the condition as the result of an acute but not necessarily destructive lesion in the external portion of the homolateral posterior column of the upper cervical cord. It occurred in 40 per cent. of our cases, i.e., in nearly the same proportion as two of the conditions which are commonly described as "typical" of the disease, namely, spastic paraplegia and intention tremor.

Evidence of interference with the pyramidal system of fibres was obtained in a large majority of our cases. Thus an extensor plantar response was present in 91·4 per cent., bilateral in 77·1 per cent., and unilateral in 14·3 per cent., while the abdominal reflexes were absent on both sides in 77·1 per cent. Spastic weakness of both legs sufficient to warrant the description of spastic paraplegia obtained in less than half the cases (45·7 per cent.). In only two cases was an atrophic paralysis present (Cases II and XIII).

Inco-ordination of movement, apart from that due to the falling out of afferent impulses from deep structures, was present in approximately half the cases. In a disease such as disseminated sclerosis where motor and sensory tracts are prone to be affected simultaneously at different levels of the cerebro-spinal axis, it is not always easy to identify precisely what elements of disordered co-ordination are due respectively to paresis, to loss of deep sensibility, or to interference with the cerebellar system. For example, the intentional tremor, which has so long held a prominent place in the symptomatology of the disease, is in many respects similar to, but is nevertheless not quite the same as, the rhythmical tremor associated with subthalamic lesions. The latter is a pure tremor initiated by the withdrawal of support from the affected limb, while the former is best demonstrated in the execution of some voluntary movement, and is a combination of tremor and inco-ordination. Hence we are inclined to regard the intention tremor of disseminated sclerosis as the result of

destruction of cerebello-rubral fibres and of certain other cerebellar connections in addition.

Definite evidence of cerebellar inco-ordination in one or more limbs, apart from intention tremor, was obtained in eighteen cases (51·4 per cent.). True intention tremor of one or both arms was present in 42·6 per cent., and a nodding to-and-fro tremor of the unsupported head in 37·1 per cent. Ataxic dysarthria on the other hand occurred in only one-fourth of the cases (28·6 per cent.).

Attacks of rotatory vertigo, usually sudden in onset and offset, but sometimes persisting for days or weeks at a time, were common at some period of the disease (51·4 per cent.). It is interesting to note that although tinnitus was associated with these attacks in a few cases, in no instance was any diminution of auditory acuity demonstrable at the time of examination, a fact which points to the vertigo being central, and not peripheral, in origin.

Sustained nystagmus in one direction or another, usually horizontal, was present in 74·3 per cent.

Regarding ataxic dysarthria, nystagmus, vertigo, nodding tremor of the head, intention tremor of the arms, and inco-ordination of the limbs (apart from sensory defects) as evidence of disturbance of the cerebellar system, we believe that there are good grounds for assuming that the lesions underlying these phenomena are situated in a vast majority of the cases in the brain-stem and mid-brain. The assumption is supported by the circumstance that some or all of these disturbances tend to be combined in the same individual.

For example, in eight of the ten dysarthric patients there were also present tremor of the head, intention tremor, and reeling gait, while all the cases with head tremor, except one, presented evidence of cerebellar inco-ordination in arms or legs, or in both.

Diplopia, usually transient, occurred in a third of the cases (34·3 per cent.), but evidence of involvement of cranial nerves other than the optic and oculo-motor group was rare. In no case did the pupillary reactions show any departure from the normal.

Disturbances of the visual mechanism have long been recognized as being one of the most frequent and characteristic features of disseminated sclerosis. Pallor of the optic discs was observed in nineteen cases out of thirty-three in which they were examined (57·6 per cent), bilateral in 45·5 per cent., unilateral in 12·1 per cent. The pallor sometimes involved the whole disc, but more commonly the temporal half only, and in a great many cases it was associated

with normal visual acuity. A history of sudden amaurosis in one eye with subsequent improvement in vision (retro-bulbar neuritis) was obtained in 20 per cent., while severe bilateral visual failure of sufficient degree to be termed disabling was present in only three patients (8·6 per cent.). Scotomata were present in several cases, but the details in this connection were not fully investigated.

Partial or complete loss of control over the bladder was noted in 71·4 per cent. of cases, and when not associated with a paraplegic condition was regarded as evidence of disease in the lumbo-sacral region of the cord.

As regards the mental condition, a majority of the patients were above rather than below the average intelligence. Definite loss or impairment of emotional control, with which was frequently associated an unduly sanguine temperament, was present in half the cases (51·4 per cent.), a defect which permitted the patient to regard his or her condition with indifference or even with amusement.

Loss of consciousness occurred in one patient only.

In no case were any indications observed pointing to disease of the thoracic or abdominal viscera, or to the hæmopoietic system.

The family and past histories failed to throw any light on the ætiology of the disease.

Sex.—There were twice as many females as males (65·7 to 34·3 per cent.).

Age.—The average age of onset was 24·6 years, the youngest case being 16, and the oldest 39 years old. The average age at the time of observation was 28·6 years.

Duration.—The disease had lasted four years on an average, the shortest case being three weeks, the longest fourteen years. Of the two fatal cases one lasted four years and the other thirteen months.

Summary of clinical study.—Disseminated sclerosis is a disease of young adult life, occurring in healthy individuals, characterized in a large majority of cases by an intermittent course, punctuated at irregular intervals by relatively acute exacerbations with subsequent periods of improvement and quiescence. The exacerbations consist either in the appearance of new symptoms due to lesions in fresh localities, or else in an accentuation of symptoms previously experienced, but at the time quiescent. In a small proportion of cases the disease pursues a chronic progressive course from the outset, with a gradual increase in the intensity of existing symptoms and a relative infrequency of new ones.

There is abundant clinical evidence of the random dissemination of the lesions throughout the brain, brain-stem, and cord, and of the involvement in the cord of the white matter rather than the grey. The general clinical course, especially in the case of single acute lesions, is highly suggestive of a localized acute, or subacute, inflammation. There is no evidence of thrombosis or embolism of large vessels, nor is there anything in the clinical study of our cases suggestive of meningeal inflammation or increased intracranial pressure.

Investigation of Two Fatal Cases.

Case I.—Girl, aged 19. Admitted August 12, 1913. Died November 24, 1913 (National Hospital, Queen Square). Good family and past history.

Aged 15: She suddenly lost power and feeling in the left upper extremity; the power soon returned but the hand was "useless" for nearly three months, after which it got perfectly well.

Aged 16: Sudden attack of pain in the right eye which rapidly became blind; examination revealed a retro-bulbar neuritis with profound but not complete loss of vision. Two months later the right optic disc was passing into atrophy and she could count fingers at 3 ft.

Aged 17: Numbness of the right thigh followed by weakness of the right leg and inability to walk. She was admitted to the West London Hospital where she rapidly developed incontinence of urine and fæces, with occasional retention requiring catheterization. She also had diplopia and attacks of rotatory vertigo. On discharge from hospital four moths later she walked "all over the place," and her head and her hands shook so much that she could hardly feed herself. In this condition she remained for eighteen months troubled with fleeting paræsthesiæ, diplopia, vertigo and headache.

Aged 19: One month ago the left arm became suddenly "useless," so that she could not feel what she had in her hand; this was followed a few days later by a similar condition of the right arm.

One week later a further series of symptoms developed: (i) persistent vomiting and constant vertigo, an ever present feeling of herself and her surroundings rotating, worse on movement of head and eyes, "nothing keeps still"; (ii) diplopia; (iii) inability to walk or sit up in bed; (iv) loss of emotional control; (v) retention of urine.

A week later (i) she lost all sphincter control, unconscious of "call" and of the passage of excreta; (ii) the legs became completely paralysed and numb: "I do not know that I have legs except for the pains in them;" (iii) speech became slow and stammering, and (iv) she had attacks of dyspnœa, and (v) difficulty in swallowing.

On admission (August 12, 1913).—Good physique and nutrition. Temperature 99·2° F., pulse 96. Sleepy expression (ptosis), with head turned to right.

Visceral examination negative; severe cystitis present.

Mental.—Distracted, inattentive and irrational. Wishes to die and the next moment evinces confident hopes of recovery. Lacking in reserve and emotional control.

Wassermann reaction (*blood*) negative.

Speech.—No aphasia. Cannot raise voice above a whisper, articulation jerky, slow and tremulous.

Vision.—Acuity, counts fingers at 3 ft. on right, $\frac{6}{36}$ on left. Fields: gross constriction with central scotoma in right eye, and no colour vision; moderate constriction in left. Both discs very white, edges clear, pigment disturbed; changes right more than left.

Hearing.—Normal. Constant vertigo; turning head to left causes a movement of objects from left to right.

Cranial nerves.—iii, iv, vi. Wide pupils, right more than left; the right is sluggish to direct light but reacts well consensually, left normal. Both react well to accommodation. Diplopia, paralysis of right superior rectus and left internal rectus. Ptosis, right more than left, with frontalis overaction. Constant jelly-like nystagmus with eyes at rest. On conjugate deviation both to right and left there is a slow horizontal nystagmus of large amplitude, and a rotatory nystagmus on looking up and down. v, vii, xii normal, except for slight paresis of left lower face. ix, x, xi: palate immobile, reflex absent. Dysphagia. Dysarthria.

Motor.—Head and neck: No paralysis; tremor when unsupported.

Upper limbs: (*a*) Right: General weakness, especially of extensors; normal tone, well-marked intention tremor and dysmetria. (*b*) Left: Hemiplegic attitude, spastic, movements at all joints minimal, extensors weaker than flexors, no wasting.

Trunk: Cannot move in bed. Intercostal movement below sixth rib is poor and abdominal muscles are paralysed.

Lower limbs: Right and left: Complete spastic paraplegia in extension with occasional involuntary flexor spasms.

Sensory.—(i) Subjective: Severe boring paroxysmal pains in abdomen and legs. Feels slight discomfort with distended bladder. (ii) On examination: Questionable impairment of tactile sensibility in both hands. Pain and temperature normal in upper limbs. Complete astereognosis in both hands. Profound loss of passive position and movement whole of left upper limb, considerable loss in right hand. Below the seventh rib there is an almost complete anæsthesia to all forms, both superficial and deep; a sharp prick or deep pressure sometimes gives rise to indefinite pain which cannot be localized.

Spinal column.—Healthy.

Sphincters.—Rectum: Complete incontinence; feels neither "call" nor passage. Bladder: Feels neither "call" nor passage, retention alternates with incontinence.

Reflexes.—All tendon-jerks brisk. Abdominals all absent. Extensor plantar response, right and left.

Treatment.—Bladder—wash twice daily.

Progress (September 9, 1913).—Patient has had frequent attacks of respiratory embarrassment, with very shallow movement of chest, cyanosis, restlessness, but no stridor. There is a curious irregularity of the respiratory rhythm. Increasing dysphagia, vomiting and mental deterioration.

September 15, 1913 : The upper limit of complete anæsthesia and paralysis now reaches the fourth rib ; the paraplegia is now in flexion rather than in extension. Urine is now free of pus.

September 25, 1913.—Irregular pyrexia the last few days ; trace of pus in urine. Regurgitation of fluids through nose.

October 10, 1913 : Pyrexia persists. Commencing sacral bed-sores with emaciation and cachexia. Less dysphagia, dyspnœa and vertigo.

November 18, 1913 : General condition much worse : profound wasting and cachexia with rapid extension of bed-sores.

November 24, 1913 : Died.

Pathological investigation.—The patient was lumbar punctured on September 30, 1913, and again on October 27, 1913, and the cerebrospinal fluid examined by the same methods and with the same results in each case, as follows :—

Cerebrospinal fluid quite clear ; no cells and no excess of protein ; Wassermann reaction negative. The fresh fluid was precipitated with absolute alcohol and the centrifugalized deposit stained with Giemsa and examined microscopically with negative results.

The fresh fluid was centrifugalized and the deposit added to four tubes of Noguchi's medium. Of these, two tubes remained sterile, while in the other two a long-chained streptococcus was grown which was Gram-positive and grew well in ordinary media with diffuse turbidity. The deposit from all four tubes after incubation was stained with Giemsa and examined microscopically ; in each case small granular bodies were observed, but precisely similar bodies were found in the deposit of uninoculated tubes.

No animal inoculations were carried out with the fresh cerebrospinal fluid.

A post-mortem examination of the patient's body was carried out twenty hours after death by Dr. S. A. Kinnier Wilson, who kindly gave us every facility for our investigations. Before removal of the organs cultures were made from the frontal regions of the brain on both sides and from the cord. A long-chained streptococcus was grown from each site, which acidified litmus milk and lactose, but did not affect raffinose, inulin or mannite.

Portions of the patient's brain and cord were removed with all reasonable precautions and ground in a mortar. Films of the suspension

thus obtained showed diplococci and streptococci, but no spirochætes were found by the dark-ground illumination method. The suspension was then filtered through a Doulton candle and on November 26, 1913, injected under general anæsthesia into rabbits as follows :—

Rabbit 1.—Trephined: 0·25 c.c. of suspension injected intracerebrally and 5 c.c. intraperitoneally. The animal remained in perfect health and was killed nine weeks later. Post-mortem examination revealed no disease of the internal organs. Smears were made from the cerebral and spinal membranes and stained with Giemsa with negative results. The pons and mid-brain were examined histologically and found to be healthy.

Rabbit 2.—Trephined: 0·25 c.c. of suspension injected intracerebrally. The animal remained in perfect health and was killed five months later. Histological examination revealed no disease in brain or cord.

Rabbit 3.—Injected intraperitoneally with 8 c.c. of suspension. No immediate or remote results, and the animal was well five months later.

The histological examination of the patient's brain and cord were unfortunately very incomplete, and the notes concerning it have been lost. Both brain and cord were riddled with sclerotic areas, one large one involving the whole transverse area of the cord in the upper thoracic region. In the lower half of the pons very little normal tissue remained. A perivascular round-cell infiltration was a prominent feature of the diseased areas.

Case II.—Male, aged 26. Admitted November 19, 1913. Died February 20, 1914. Family and past history unimportant. Ten months ago (January 6, 1913) he "suddenly came over sick and giddy" but was well in three days. Three weeks later the same thing happened, and his legs became so weak that he could not walk and his hands were unsteady. He was admitted to the London Hospital where he improved, and after seven weeks he could write a letter and walk without support. On discharge he began to notice transient numbness and tightness in his left leg and arm, "as if my leg would burst." These paræsthesiæ persisted, but no further symptoms developed until August, when his sight began to fail and his left eye became rapidly blind. In September his walking got much worse and he had frequent falls; he also had difficulty in voiding his urine, and towards the end of the month he developed incontinence of both bladder and rectum.

Four weeks ago his left arm became much weaker and more unsteady, his left leg almost paralysed, his speech almost unintelligable, and his memory began to fail.

On admission (November 19, 1913): Poor physique. Temperature 100·4° F., pulse 80.

Visceral examination.—Negative. Urine normal.

Wassermann reaction (*blood*).—Negative.

Mental.—Intelligent but apathetic. Normal emotional tone.

Speech.—No aphasia. Articulation slow, scanning, tremulous and ataxic.

Vision.—Acuity: Left $\frac{6}{60}$, right $\frac{6}{18}$. Left optic disc very pale, edges fluffy, some disturbance of retinal pigment; consecutive atrophy. Right optic disc pale, especially on temporal side; primary atrophy.

Hearing.—Normal. No vertigo now, or tinnitus.

Cranial nerves.—Normal except for weakness of right lower face, and horizontal nystagmus of wide range on looking to right and left.

Motor.—Head and neck: Nodding tremor of unsupported head.

Upper limbs: (*a*) Right, power everywhere subnormal, tone a little increased, well-marked intention tremor and slight sensory ataxia. (*b*) Left, muscles of forearm and hand weak and wasted, with diminution of faradic irritability, extreme intention tremor and sensory ataxia.

Trunk: Cannot sit up without use of hands.

Lower limbs: Both weak and spastic, left more than right; all flexors weaker than extensors. Legs in extension with occasional flexor spasms in left leg. Musculature poor. Legs tremulous.

Sensory.—Profound loss to pin-prick and temperature to right of mid-line below umbilicus. Tactile sensibility greatly impaired whole of left lower limb. Vibration completely abolished in lower limbs and pelvis and much impaired in hands (left more than right).

Compasses: No threshold obtainable in palms or soles.

Complete astereognosis in both hands.

Passive posture and movement: Profound loss distally in upper limbs (left more than right), considerable loss in lower limbs (left more than right).

Spinal column.—No evidence of disease.

Sphincters.—Complete incontinence of urine and fæces; feels neither call nor passage.

Gait.—Cannot stand alone; very tremulous; no control over legs.

Reflexes.—Left supinator jerk absent, all other jerks very brisk. All abdominals absent. Extensor plantar response, right and left.

Progress (December 18, 1913).—Condition deteriorating. Dysarthria is now so profound that he is barely intelligible. Mild irregular pyrexia; bed-sore developing over sacrum; urine normal.

January 15, 1914: Legs are now completely paralysed; paraplegia in flexion.

February 13, 1914: Whole of left face and left side of palate paralysed. Dysphagia has developed with regurgitation of fluids through nose. Complete loss of upward movement of both eyes.

Still slight irregular pyrexia. Bed-sore has increased, but is not deep and is fairly clean. No cystitis.

February 20, 1914: Died.

Pathological investigation.—The patient was lumbar punctured on December 1, 1913, and on December 18, 1913. The cerebrospinal fluid was quite clear, there was no excess of globulin, and the Wassermann reaction was negative. In the fluid from the first puncture no cells were found, while in the second there was a very slight excess of small lymphocytes.

On December 1, 1913, two rabbits were injected intraperitoneally, one with 15 c.c. and the other with 10 c.c. of the patient's unheated blood-serum without immediate effect. Two days later fresh cerebrospinal fluid was obtained from Case III (from which a streptococcus had been previously grown; *vide infra*), and 0·25 c.c. of the centrifugalized deposit was injected intracerebrally into both rabbits. Both animals remained well; one was killed six weeks later and no histological or naked-eye changes were found in the brain or cord, the other was alive and well three months afterwards.

Cultures of the fresh cerebrospinal fluid of Case II, both aerobic and anaerobic, remained sterile, and examination of the stained centrifugalized deposit was negative. On December 18, 1913, two rabbits were injected intracerebrally with 0·5 c.c. of the centrifugalized deposit of fresh cerebrospinal fluid. Both remained well; they were killed three and four months later respectively and no lesions, naked-eye or microscopic, were found in the brain or cord.

Post-mortem examination of Case II (six hours after death).—No naked-eye evidence of disease in internal viscera. Numerous sclerotic foci were present in the cerebral cortex, basal ganglia, periventricular tissue, pons, medulla, and spinal cord (especially in cervical region); none were visible in the mid-brain. No naked-eye evidence of meningitis. Portions of the brain and cord were ground up in a mortar with physiological saline, filtered through a Berkefeld V-candle, and three hours after removal of the tissue from the body 0·5 c.c. of this filtrate was injected intracerebrally into a rabbit and 4 c.c. intraperitoneally. Three days later another 7 c.c. of filtrate was injected intraperitoneally. The animal remained well and was killed four and a half months later; nothing abnormal, naked-eye or histologically, was found in the brain or cord.

Histological.—Both kidneys showed chronic tubal change.

Sections of brain and cord stained by the Weigert-Pal method gave the following results:—

Numerous small foci of myelin sheath destruction were found scattered throughout the cerebral hemispheres both in the cortex and

adjacent white matter, especially in the anterior half of the brain. Extensive sclerotic patches were present in the basal ganglia and in the periventricular regions.

Subthalamic region: The red nucleus on either side was involved in a patch of sclerosis.

Pons: numerous sclerotic foci were scattered throughout the pons, especially round the fourth ventricle, in the region of the right middle cerebellar peduncle, and both facial nuclei.

Medulla: numerous foci were present, right more than left; the region of the right restiform body was occupied by an area of particularly dense sclerosis.

Spinal cord: (i) Upper cervical: sclerotic areas occupied the central and external portions of both posterior columns, and a large focus involved the left antero-lateral region and anterior horn.

(ii) Lower cervical: sclerotic foci present in both anterior columns, in the crossed pyramidal tract on the right side, and in the posterior columns on both sides.

(iii) Mid-thoracic cord: both posterior columns involved throughout in an area of sclerosis. On the right side the lateral column, and on the left the antero-lateral column were occupied by areas of dense sclerosis, which invaded the anterior horn on the left side.

(iv) Lumbar: whole transverse area, except the anterior columns, occupied by dense sclerosis.

Cerebellum, spinal roots, and ganglia—not examined.

Sections stained with Giemsa showed that the patches of sclerosis were not all sharply defined from the normal tissue. This lack of definition was greatest in the areas which appeared to be fresh; in these, especially in the cortex and brain-stem and to a very much smaller extent in the cord, the nerve cells showed chromatolysis and indistinctness of outline, and glia-cell proliferation was a prominent feature. In the older areas the gliosis was more intense and characterized by proliferation of glia fibrils.

In the leptomeninges and throughout the brain and cord a perivascular infiltration with round cells and endothelial cells was present. This change was particularly noticeable in the fresh areas of sclerosis, but was not entirely absent in areas which were of long standing.

Investigations carried out in Thirty-three Non-Fatal Cases.[1]

(1) *Examination of Cerebrospinal Fluid.*

The examination comprised the following points :—

(*a*) Cytology (all cases).

(*b*) Globulin reaction (all cases).

(*c*) Wassermann reaction (all cases).

(*d*) Microscopical examination of deposit obtained by precipitating the fresh cerebrospinal fluid with absolute alcohol, stained with Giemsa (all cases).

(*e*) Anaerobic cultivation (27 cases).

(*f*) Microscopical examination of deposit in Noguchi tubes after inoculation with cerebrospinal fluid and incubation (25 cases).

(*g*) Aerobic cultivation (19 cases).

The colloidal-gold reaction was not tested in any case.

The results obtained were as follows :—

(*a*) No excess of cells was obtained in any of the 33 cases. Occasionally one lymphocyte was found in the centrifugalized deposit but in the vast majority of cases no cells of any kind were present. It will be remembered that in one specimen of cerebrospinal fluid from Case II a slight lymphocytosis was present.

(*b*) Globulin reaction (tested by half saturation with ammonium sulphate) ; a positive reaction was present in Case XXXIV, a very slight reaction in Cases XXVII, XXXI and XXXV, and a negative reaction in the remaining 29 cases.

(*c*) The Wassermann reaction was negative in every case, both in the blood and the cerebrospinal fluid.

(*d*) Deposit obtained by precipitation with absolute alcohol : in Case VIII examination of the deposit stained with Giemsa revealed numerous small bodies resembling cocci. [In two cases (XXXIV and XXXV) small granules resembling fat were observed by the dark-ground illumination method ; similar bodies have been observed in the cerebrospinal fluid and brain emulsions of cases of encephalitis lethargica and uræmia and are probably fragments of altered myelin. With these exceptions this method yielded negative results.]

(*e*) Anaerobic cultivation. The following media were used :—

(i) Noguchi's fluid medium (ascitic fluid + fresh rabbit's kidney, in 23 cases (III to XXI, XXX and XXXI, XXXIV and XXXV).

[1] For clinical details of cases *vide* Appendix.

(ii) Ascitic fluid + human red cells in three cases (XXIII to XXV).

(iii) Noguchi solid medium (agar + rabbit's kidney) in two cases (XXX and XXXI).

(iv) Ascitic fluid only in Case XXII.

In twenty-one cases all the inoculated tubes remained sterile. In three Cases (IV, XIX, XX) a diphtheroid bacillus was grown; in Cases IV and XIX the same bacillus also grew in aerobic cultures. The bacillus grown anaerobically in Case XX was non-pathogenic to guinea-pigs.

Case III, from which a streptococcus was grown, merits a more detailed description. The patient was a girl, aged 18, and the illness dated back to just over a year previously when she was suddenly seized with pain behind the eyes and failure of vision. The vision improved somewhat, but in the course of the next four months she developed successively weakness of the trunk muscles, tremor of the left arm, loss of sphincter control, weakness of the right leg and then of the left. At the time of our investigations she was rather rapidly getting worse and becoming more paralysed below the waist; there were however no acute exacerbations, no bed-sores, cystitis, or pyrexia.

Lumbar puncture was performed on three occasions as follows:—

(i) September 30, 1913. The centrifugalized deposit of cerebrospinal fluid was added to four Noguchi tubes, of which two remained sterile, while in the other two a pure growth of a Gram-positive, long-chained streptococcus appeared, which grew well in ordinary media with diffuse turbidity. Three rabbits were then inoculated intracerebrally with 0·5 c.c. of a twenty-four hours' growth of the streptococcus. Two days later two animals were dead, and examination revealed frontal abscesses, the pus from which contained long-chained streptococci in large numbers. The third animal was ill two days after inoculation, but recovered rapidly. It was killed eight weeks later, and post-mortem examination revealed nothing abnormal in brain, cord, or meninges, and cultures from the meninges remained sterile.

Intraspinal injection into a rabbit of 1·5 c.c. of fresh cerebrospinal fluid was followed by no immediate or remote effects, and histological examination of the animal's cord ten weeks later revealed nothing abnormal.

(ii) October 27, 1913. Patient was again lumbar punctured, but on this occasion all anaerobic and aerobic media remained sterile.

(iii) December 3, 1913. Lumbar punctured. Two rabbits, both of which had two days previously received an intraperitoneal injection of the unheated blood serum of Case II (fatal case), were injected intracerebrally with 0·25 c.c. of the centrifugalized deposit of cerebrospinal fluid. In neither animal was any effect produced.

The streptococcus grown from the first sample of cerebrospinal fluid in this case resembled that grown post mortem from the brain of Case I in both its morphological and cultural characteristics.

(*f*) Examination of Noguchi deposit. Films were stained with Giemsa and searched for parasites with negative results. In several cases small granular amorphous bodies were found, which were particularly numerous in Case XXI. In this case 10 c.c. of the Noguchi fluid were injected intraperitoneally into a rabbit which two weeks previously had received 2·5 c.c. of the patient's cerebrospinal fluid along the course of one sciatic nerve plus 5 c.c. of the patient's blood serum intraperitoneally. The animal however remained well.

Similar granular bodies were frequently found in the deposit from Noguchi tubes which had not been inoculated with cerebrospinal fluid.

(*g*) Aerobic cultivations: various media were used, litmus milk, inulin, inulin plus serum, Besredka with and without human red cells, &c.

In Cases IV and XIX a diphtheroid bacillus was grown, and from Case XIII a pure culture of *Staphylococcus albus* was obtained, which we regard as a contamination.

With these exceptions the cultures all remained sterile.

(2) *Animal Experiments* (*Thirteen Cases*).

Rabbits were employed in every case, and in a few instances guinea-pigs in addition. All animal experiments were conducted under general anæsthesia and with strict aseptic precautions. We consider the use of a general anæsthetic important in order to obviate the traumatic palsies, especially of the hind limbs, to which rabbits are particularly susceptible; these palsies which occur in animals improperly treated have been a fruitful source of error in the past.

To the experiments in connection with the two fatal cases in which intracerebral injections of suspensions of cerebral and cord tissue were effected, reference has already been made. The intracerebral route was also used in Cases III, XXVII and XXVIII, the material injected in each case being 0·25 c.c. of the centrifugalized deposit of fresh cerebrospinal fluid.

Injections were made of from 2·5 to 5 c.c. of fresh cerebrospinal fluid along the course of the sciatic nerve in Cases XIX, XX, XXI, XXVI and XXVIII. In the first three of these cases intraperitoneal injections of from 5 to 10 c.c. of the patient's unheated blood serum were given simultaneously, and in Case XXVIII an intracerebral injection (0·25 c.c.) of centrifugalized cerebrospinal fluid.

In Case III an intrathecal injection of 1·5 c.c. cerebrospinal fluid was given, in Case XXV an intravenous injection of 2 c.c., and in Case XXIX an intratesticular injection of 3 c.c.

In none of the above cases were the injections followed by any immediate or remote effects; all the animals remained in good health, and in those that were killed at some subsequent period no lesions, naked-eye or histological, were found in the internal viscera, meninges, brain or spinal cord.

In Cases XXXIV and XXXV the intra-ocular route was adopted. In each case a rabbit was inoculated with 0·25 c.c. of centrifugalized deposit of cerebrospinal fluid withdrawn from the patient two hours previously. Six days later the rabbit inoculated with cerebrospinal fluid from Case XXXIV was disinclined to move and its hind limbs were stiff. The following day the weakness and stiffness of the hind limbs were more obvious, the animal responded slowly when pricked, its gait was laboured and there was some "shivering" of the muscles in the lumbar region. Two days later the animal had completely recovered and is alive and well at the time of writing. Six days after injection the animal from Case XXXV showed an exactly similar condition, but the weakness of the hind limbs was rather more pronounced, and the limbs were actually dragged along without being definitely paralysed. After persisting for two or three days the disability completely disappeared and the animal has remained well ever since.

Two guinea-pigs injected intraperitoneally with fresh cerebrospinal fluid from Cases XXXIV and XXXV remained well.

Summary of animal experiments and bacteriological investigations.—It will be seen from the above experiments that in spite of a variety of routes employed for our injections we failed to produce in rabbits any condition of ill-health, with the exception of a transient stiffness of the hind legs in two animals, by inoculating them with the fresh cerebrospinal fluid and blood-serum of patients suffering from disseminated sclerosis, or by inoculating them with fresh, filtered suspensions of the brain and cord of patients recently dead of the isease.

In other words we have entirely failed in our attempts to transmit disseminated sclerosis to animals.

As regards our bacteriological findings a long-chained streptococcus was grown in two cases, from the cerebrospinal fluid of Case III, and from the cerebrospinal fluid during life and from the cerebral hemispheres after death in Case I. In the latter case (Case I) owing to cystitis and bed sores the patient had been in a profoundly septic and cachectic condition for six weeks prior to her death, and the bacteriological result may reasonably be thus explained. Case III however had neither cystitis, bed-sores, nor pyrexia, and the presence in the cerebrospinal fluid of a streptococcus, cultures of which on intracerebral injection into rabbits caused death with the formation of frontal abscesses, was not traced to a septic focus. At the same time, while unable to advance any adequate explanation of the bacteriological finding in this case, we are by no means inclined to regard it as of any significance with reference to the pathogenesis of disseminated sclerosis.

Review of Recent Work on the Pathogenesis of Disseminated Sclerosis.

(1) *Histological. Morbid Anatomy.*

Two exhaustive investigations of the morbid anatomy of the disease have appeared within the past few years.

(*a*) Siemerling and Raecke in 1914 published the results of detailed histological examination of eight cases together with a clinical review of sixty others. They consider that the primary and essential feature of the pathological process is a localized destruction of nervous tissue, the process being closely related to changes in the vessels, which show definite inflammatory phenomena, viz., congestion, perivascular infiltration with small lymphocytes, polymorphs and plasma cells, and in many cases capillary hæmorrhages.

They describe the early changes, best studied in the small, circumscribed patches ("Herde") in the brain, as being characterized by a focal destruction of axis cylinder fibres, with which is associated a considerable destruction of the medullary sheaths. The foci tend to coalesce and the medullary sheath destruction becomes more and more complete. In the cerebral cortex and periventricular region the lesions are grouped according to the distribution of terminal arteries and resemble infarcts, the cortical foci usually having a pial vessel in their centre. Immediately consequent on the destruction of nervous tissue

a marked proliferation of the glia occurs, which acts as scar tissue and helps to protect the tissues from further damage. The early stages of the glia reaction are characterized by a richness of glia nuclei, which later on are reduced in number to be replaced by a thick network of glia fibrils. Associated with the myelin sheath destruction numerous scavenger cells make their appearance, arising mainly from the neuroglia and to a less extent from the blood elements. These cells, which effect the transportation of the products of destruction, are chiefly compound granular cells, and are most numerous in fresh lesions and in the lymphatic spaces outside the vessels. They are never as numerous as in cases of softening due to arterio-sclerosis, and are much scarcer in the cortex than in the white matter, since myelin destruction is negligible in extent in the former as compared with the latter. These cells contain a large, deeply-staining, round nucleus and their protoplasm is frequently packed with fatty granules staining orange-red with scharlach.

Owing to the excessive glia reaction neighbouring foci tend to be bound together in a common sclerotic plaque. Moreover, the individual foci do not pass directly over into normal tissue, but are surrounded by a broad intermediate zone characterized by a pronounced increase of glia nuclei. In these zones the authors observed a tendency to recurrence of capillary hæmorrhages, while the presence in them of plasma cells was regarded as indicating a lighting up of the active process.

Secondary degenerations, as a result of axis cylinder destruction, were found in distant parts of the nervous system, and mingling with the essential focal lesions produce a complicated and confusing histological picture. While, however, axis cylinder destruction is the first stage in the morbid process and is demonstrable before the medullary sheath is affected, the authors admit that the latter change eventually exceeds the former, and they do not entirely oppose the long-accepted view that the axis cylinders remain relatively intact.

The ganglion cells, although more resistant than the nerve-fibres, also show changes when involved in the primary lesions, but the changes were limited to the focal lesions and were never systemic as in general paralysis.

Occasionally the glia over wide areas, and altogether outside the essential focal lesions, undergoes definite proliferation with an abnormal richness of both nuclei and fibrils and a few pathological spider cells. A section of such an area would certainly give the impression of a

primary glia proliferation, but the authors are of opinion that there are in reality no grounds for postulating a primary gliosis, and they explain the appearances by a leakage from the diseased areas into the periphery of a virus or toxin in a concentration too weak to evoke a focal inflammatory lesion, but sufficiently strong to give rise to a diffuse glia reaction.

The peripheral nerves showed inconstant, non-specific, neuritic changes, and the typical sclerotic plaques were strictly limited to the central nervous system and to the glial portions of the cranial nerves and nerve-roots. In correspondence with the inflammatory nature of the whole process meningitic changes were constantly present, e.g., patchy peri-vascular round-celled infiltrations of the pia-arachnoid; plasma cells were also observed in a few cases.

The cerebrospinal fluid was examined in both fatal and non-fatal cases, and is described as showing "usually a slight lymphocytosis and slight increase of globulin." Bacteriological investigation was negative.

The authors consider that the whole anatomical picture is in complete harmony with the clinical one, viz., acute lesions giving rise to acute symptoms which tend to clear up. They conclude that disseminated sclerosis is infective or toxic in origin, that the virus or toxin reaches the central nervous system through the blood-vessels, and they regard the presence of plasma cells as strong evidence in favour of an infective process and most probably a parasitic one.

(*b*) Dawson examined nine cases histologically and came to much the same conclusions as Siemerling and Raecke, although differing somewhat in his interpretation of the histological details. He describes six stages in the development of the essential lesion, viz.: (1) A commencing reaction of all the tissue components and degeneration of the myelin sheath. (2) Glia cell proliferation and commencing fat granule cell formation. (3) "Fat granule cell myelitis." (4) Commencing glia fibril formation. (5) Advancing sclerosis, and (6) Complete sclerosis.

He regards the primary degeneration of the myelin sheath as the most constant and uniform feature of the histological picture, and considers that the relative persistence of axis cylinders and ganglion cells is abundantly confirmed. Secondary degeneration occurs, but affects scattered fibres and not tracts in their entirety.

He traces the development of the essential lesions from their

earliest stages to the final development of the dense sclerotic areas visible to the naked eye and scattered throughout both brain and cord, and he finds areas of recent and of old disease in the same patient. Almost every diseased area shows evidence of an advancing process; they are either wholly "early" or show a peripheral advancing zone round a condensed centre, giving the impression that the primary process has never quite died down but is gradually extending peripherally.

According to Dawson vascular phenomena are not prominent in the earliest stages; "in its diffusion through the vessel wall the noxa causes no recognizable primary alteration, but there is probably an abnormal permeability and increased transudation of toxic lymph." The first vascular changes are found at, or just after, the stage of "fat granule cell myelitis," and are secondary to the resorption of the fatty products of destruction; this in time is followed by a proliferation of all the cell elements of the adventitia, and later by a modified infiltration of small round cells and a few plasma cells. Finally as the tissue condenses the vessel walls become thickened and undergo hyaline degeneration.

The cranial nerves and spinal roots are frequently involved in the glial portion of their extra-medullary course, but the meninges show no constant or specific changes.

While not attempting to explain the irregular distribution of the lesions, Dawson considers that the sites of predilection are "probably related (*a*) to the vessels; to the terminal ramifications of end-arteries, e.g., on the ventricular surfaces, and to the points where vessels break up, e.g., in the transition zone between white and grey matter both in the central and cortical grey matter, and (*b*) to areas where much glia is normally present, e.g., the peri-ventricular and peri-central tissue, the optic chiasma, the postero-median and para-median septa, the marginal glia zone, and the perivascular glia layer."

After discussing the somewhat different views as to what should be accepted as the histological criteria of "inflammation" when it affects the central nervous system, he concludes by regarding disseminated sclerosis as a subacute inflammatory disease, i.e., a localized, disseminated, subacute encephalo-myelitis which terminates in areas of actual and complete sclerosis, the causal agent being probably a soluble toxin which is conveyed to the nervous system by the blood-stream. He further points out that the remissions and relapses so characteristic of the disease "necessitate the assumption of the latent

presence of the morbid agent in the body, or, if this is an autogenous toxin, either of its intermittent evolution or of its accumulation from deficient elimination."

Reviewing the views propounded respectively by Siemerling and Raecke and by Dawson, while both sets of investigators regard the histological changes as inflammatory in character, the former look upon the process as acute, while Dawson calls them subacute.

Our own histological investigations are admittedly quite incomplete; but as far as they go they strongly support the view which regards the process underlying disseminated sclerosis as inflammatory in nature; in fact the most conspicuous feature of the sections in Case II was the well-marked perivascular infiltration with round cells and endothelial cells, similar to that met with in cases of encephalitis lethargica, although perhaps not quite so intense. On general grounds we have some difficulty in accepting Dawson's statement that the perivascular changes are absent in the earliest stages of the process, and only appear subsequent to, and as a consequence of, the absorption of the products of myelin destruction and the gradual condensation of the tissues. In Case II these changes were a prominent feature of areas where sclerosis was not yet apparent, and we favour as being more in conformity with the principles of general pathology the interpretation of Siemerling and Raecke, who regard the perivascular reaction as important evidence of the frankly inflammatory nature of the early stages of the morbid process.

(2) *Experimental.*

(*a*) Bullock in 1913 claimed to have transmitted the disease to rabbits by subcutaneous injection of the cerebrospinal fluid of a patient suffering from disseminated sclerosis. Ten rabbits in all were injected; of these four died without paralysis, two remained well, and two became paralysed in the hind limbs three weeks later and then recovered. One animal, twelve days after receiving 2 c.c. of unfiltered cerebrospinal fluid along the course of one sciatic nerve, became paralysed in the hind limbs; killed four days later, an intense cystitis was found, together with some fragmentation of the myelin sheaths in the white matter of the spinal cord, but no Marchi changes. The other animal three weeks after an injection of 1·5 c.c. filtered cerebrospinal fluid became weak in the hind limbs, but afterwards improved and was left with a slight spastic paresis. Six weeks later it was killed, and large areas of degeneration were found throughout the cord in Weigert-Pal

and in Marchi preparations. The verbal description of, and the figures which claim to illustrate, these changes are in our opinion unconvincing, and we are quite unable to accept the author's statement that the changes represent "a complete reproduction of the appearances found in the human subject"

(*b*) Siemerling and Raecke injected rabbits and two apes (*Macacus rhesus*) with cerebrospinal fluid of patients suffering from disseminated sclerosis, with negative results.

(*c*) Kuhn and Steiner injected blood and (or) cerebrospinal fluid of four disseminated sclerosis patients into guinea-pigs and rabbits. From their brief description the diagnosis in Cases III and IV is, in our opinion, open to question. Six guinea-pigs were inoculated intraperitoneally with 1 c.c. of a mixture of blood and cerebrospinal fluid from Case III, of which four developed paralysis and two remained well. Of three guinea-pigs injected with 1 c.c. of blood diluted 1 in 5 from Case IV, one remained well, one was ill four weeks later and recovered, and one became ill five weeks later and died after nine days. Blood-stained cerebrospinal fluid (0·2 to 0·4 c.c.) from Case I, diluted 1 in 4, was injected intraperitoneally into ten guinea-pigs and five rabbits. Three of the guinea-pigs showed paralytic phenomena a few days later, one became affected seven weeks later, two died of epidemic disease, and four remained well. The rabbits showed transient phenomena, not definitely paralytic, but otherwise the results in them were negative. A little later blood-free cerebrospinal fluid from Case I was injected intraperitoneally into five guinea-pigs with negative results, and 1 c.c. of defibrinated blood into another set of five guinea-pigs, of which one died with paralytic phenomena eighteen days later, and the other four of epidemic disease. Intraperitoneal injections of cerebrospinal fluid from Case II into ten guinea-pigs were without result, while similar injections of 1 c.c. of blood diluted 1 in 3 were followed six or seven weeks later by paralytic phenomena in two out of three animals. Intra-ocular injections of cerebrospinal fluid (0·2 to 0·5 c.c.) from Case II were followed by paralytic phenomena in two out of five rabbits.

These authors also claim to have transmitted the disease through a series of four guinea-pigs and later of two rabbits, using for the first injection material obtained from previously inoculated animals who had developed paralytic phenomena. The exact nature of the material used and the route employed for the injections are not stated.

Control experiments (fifteen rabbits and thirty guinea pigs) were negative.

All the affected animals showed symptoms in from three days to three months after injection (average six to seven weeks); they moved about less, sat hunched up, the hind legs became stiff and weak, and total paralysis preceded death.

Post-mortem examination of the paralysed animals revealed no naked-eye lesions in the internal viscera or nervous system, and at the time of publication the histological examination had not been completed.

Delicate, slender spirochætes, resembling those found in epidemic jaundice, were demonstrated during life and after death in the heart's blood of several of the affected animals, and also in the blood-vessels of the liver.

If we analyse these experiments it will be evident that in the absence of any histological examination of the brain and cord of the affected animals there can be no justification for assuming that the animals had developed disseminated sclerosis. This being so we are justified still less in assuming that the finding of spirochætes in the heart's blood and liver is in any way related to the origin of disseminated sclerosis, whatever may have been its relation to the morbid condition from which the experimental animals were suffering.

(*d*) Simons, using sterile cerebrospinal fluid of a patient with disseminated sclerosis, kept at 0° C. for ten days, inoculated nine rabbits, of which seven remained well. One animal, which had received 3 c.c. in one sciatic region, nine days later developed paralysis of the hind legs and died totally paralysed on the nineteenth day; no naked-eye lesions were found in meninges, brain, or cord. The other animal developed paresis of the hind limbs on the fifth day, which persisted; the animal was killed on the fourteenth day but nothing abnormal was found post mortem.

(*e*) Marinesco inoculated the cerebrospinal fluid of two patients with disseminated sclerosis into six guinea-pigs, of which four were unaffected. The other two, who had received 1 c.c. intracerebrally, presented three or four days later difficulty in moving about. In the fluid obtained by puncture of the fourth ventricle of these animals spirochætes were found similar to those described by Kuhn and Steiner. This fluid injected into guinea-pigs, and a repetition of the experiments *de novo*, all gave negative results.

(*f*) Steiner, in March, 1917, injected 1 c.c. of cerebrospinal fluid from Case I of Kuhn and Steiner's series into an ape (*Macacus rhesus*). The animal remained well until February, 1918, when some awkwardness and weakness of the hind limbs was noticed by the attendant.

This passed off to reappear in June when the limbs were observed to be weak and spastic. The condition persisted and a month later the animal was killed. At the post-mortem examination nothing abnormal was found in the viscera, but focal lesions were visible to the naked-eye scattered irregularly throughout both cerebral hemispheres. Histologically these foci were found to consist of fairly sharply outlined areas characterized by a patchy destruction of medullary sheaths; compound granular cells were present in large numbers, both these and the glia cells containing granules which stained red with scharlach. Proliferation of the glia cells was a prominent feature, and in the periphery of the lesions numerous multi-nucleated glia cells with abundant protoplasm were present, the cells showing a definite tendency to form fibrils. The axis cylinders were relatively intact and there were no vascular changes. The spinal cord apparently was not examined.

Steiner considers that the lesions found are indistinguishable from those of human disseminated sclerosis, but utters a note of warning against drawing hasty conclusions.

(*g*) Rothfeld, Freund, and Hornowski inoculated by a variety of routes guinea-pigs and rabbits with blood and cerebrospinal fluid from four undoubted cases of disseminated sclerosis. The blood of the experimental animals was examined for spirochætes almost daily, minute histological examination was made of the brain and cord, and emulsions of brain and cord from inoculated animals was injected into a further series. In no case were spirochætes found, and the authors express the opinion that the spirochæte found by Kuhn and Steiner was an accidental infection transmitted from animal to animal. Many of the inoculated animals died, but the cause of death in the vast majority of cases was either coccidiosis or tuberculosis. In one animal slight neuroglia changes were found, but precisely similar changes were observed in animals with coccidiosis who had received no injections. In all other respects histological examination of the nervous system was negative. Some of the animals appeared to be ill for a day or two after inoculation with cerebrospinal fluid, the fluid being probably toxic in some way. In no case, however, did the authors succeed in transmitting to guinea-pigs or rabbits any condition resembling human disseminated sclerosis.

(3) *The Finding of Spirochætes in the Brains of Patients dead of Disseminated Sclerosis.*

(*a*) Siemerling was the first to publish a positive result. In the brain of a patient with disseminated sclerosis who died of erysipelas, examined two hours after death, numerous focal lesions were present; minute pieces from the diseased areas were removed and examined by the dark-ground illumination method. In two preparations living spirochætes were found, similar to those discovered by Kuhn and Steiner, but attempts to stain them in sections did not succeed.

(*b*) Büscher reported the finding of spirochætes by the dark-ground illumination method in the brain of a chronic case of disseminated sclerosis; the parasites exhibited undulatory movements from fifteen to thirty-nine hours after death. They could not however be detected in hardened sections of the brain in ordinary silver preparations.

(*c*) Schuster describes the case of a boy which he regards as a combination of juvenile general paralysis and disseminated sclerosis. Mental symptoms were a prominent feature of the illness and in the later stages the pupillary reactions to light became sluggish. At the autopsy the brain was found to be atrophic, the leptomeninges thickened, and the lateral ventricles dilated. The peri-vascular spaces were packed with small lymphocytes and plasma cells, and there were numerous areas of myelin sheath destruction, especially in the cerebral white matter. Spirochætes, hardly distinguishable from the *Spirochæta pallida*, were found in the brain in silver preparations of hardened material. The Wassermann reaction was positive during life in both blood and cerebrospinal fluid and the case appears to us, but not apparently to the author, to be of no decisive importance in relation to the pathogenesis of disseminated sclerosis.

General Discussion and Summary.

In attempting to elucidate the pathology of any obscure disease there is a natural temptation to make comparisons between the disease in question and certain other apparently similar conditions the cause and nature of which are understood. In the case of disseminated sclerosis, however, it is difficult if not impossible to conjure up any analogous condition, nor, even if we could, would such analogy be decisive. In view of the suggested spirochætal origin of the disease one naturally compares it with syphilis of the nervous system, to which it bears certain resemblances in its clinical course and manifestations;

it differs however from syphilis in two important particulars, first by being rigidly confined to the nervous system, and secondly by the irregular, patchy distribution of the essential lesions in marked contrast to such systemic processes as tabes dorsalis and general paralysis.

The past and family histories of our patients, so valuable in the etiological studies of other diseases, tell us nothing as to the origin of disseminated sclerosis. It is neither familial nor epidemic, although its rarity in certain countries (it is said to be unknown in Japan) points rather to some extraneous factor.

The pathogenesis of any disease can be considered from two points of view, first the nature of the morbid process, and secondly its origin. As to the nature of the morbid process underlying disseminated sclerosis two views are held. According to the first or exogenous theory, supported by a majority of observers, the process is set up by some irritant distributed through the nervous system by the blood-stream, and is inflammatory or toxi-infective in character. The second or endogenous theory, advocated particularly by Strümpell and Müller, regards the disease as the result of some developmental or congenital defect of the neuroglia, which is thus rendered more liable to be affected by irritation than is normally the case; in other words the disease is a primary gliosis.

There would appear to be an insuperable obstacle to the acceptance of the second or endogenous theory, namely, the clinical character of the disease, since it is hardly conceivable that a condition so frequently characterized by an acute or subacute onset and a clinical course punctuated at irregular intervals with exacerbations and remissions can be ascribed to a congenital or developmental defect. Müller has attempted to overcome this difficulty first by postulating certain external factors as "agents provocateurs" acting on a neuroglia which is congenitally vulnerable and susceptible, but exactly what these factors are he is unable to say. Secondly, and more important, he attempts to draw a distinction between true disseminated sclerosis and secondary disseminated sclerosis. According to Müller the only true disseminated sclerosis is the chronic progressive case originally described by Charcot, which one can perhaps accept as being clinically intelligible as a primary progressive gliosis, whereas the cases with acute symptoms and a discontinuous clinical course, with which this paper mainly deals, he excludes altogether from the category of true disseminated sclerosis, and regards them as something else which he calls "disseminated encephalo-myelitis."

We have now to inquire whether this differentiation advocated by Müller is warranted by the clinical and histological facts at our disposal. Clinically while it is useful to recognize the chronic progressive and the subacute remittent type of case, the distinction must not be pushed too far, since cases not infrequently occur which are intermediate between the two and serve to link the one type with the other. Secondly there is a definite tendency for the remittent type in its later stages to lose its subacute characters and to take on a chronic progressive course. Thirdly, just as the chronic type of case progresses downhill by an increase in intensity of the same symptoms which initiated the disease, so too one frequently observes in the exacerbations of the remitting type a tendency to the reappearance of symptoms which have already been experienced, so that for example the same arm again becomes astereognostic, the same paræsthesiæ are again complained of in the same part of the body, the same leg again begins to drag, and so on. Fourthly, the localization of the lesions as judged by the symptoms and physical signs is roughly similar in the two types; in both, for example, there is the same curious liability to affection of the optic nerve and tract. Consequently in spite of differences in the chronological order of individual symptoms, the symptomatology of the later stages is very similar in both types, and although the individuality of no disease can be established by its symptomatology alone, this factor nevertheless must be given due weight in an attempt to determine the entity of any morbid condition. Finally, in both types there is the same absence of any obvious etiological factor and the same age incidence.

Histologically no essential differences have been shown to exist between the chronic progressive and the remittent type of case. In the former it is true that a progressive gliosis is perhaps the most prominent feature, and that areas of diffuse gliosis occur apparently unrelated to areas of tissue-destruction. Nevertheless it has been abundantly demonstrated that even in these long-standing cases areas of tissue destruction are present in which the changes are of recent date and associated with an inflammatory reaction. While therefore gliosis may be the predominating feature of the chronic case, it must be recognized that the differences in the histological picture in the two types of cases are differences of degree only; in both types the essential lesions are of the same kind, although variations in the age and intensity of the morbid process undoubtedly occur

Neither from the clinical nor histological standpoint therefore can we find adequate grounds for distinguishing between *true* and *secondary* disseminated sclerosis as advocated by Müller, and we believe that the two clinical types, namely, the chronic progressive and the subacute remittent, represent variations of the same pathological process. If this conclusion is correct, the endogenous theory automatically becomes untenable, since it is impossible to explain the remittent type with its sudden exacerbations and long remissions on an endogenous basis.

The question therefore arises: Can a disease so variable in its clinical manifestations, and running sometimes a chronic progressive and at others a subacute remittent course, be satisfactorily explained on a single pathogenetic basis? We believe that this is possible provided one assumes the presence of a morbid agent acting with varying degrees of intensity over a considerable period, and since variations in intensity are characteristic of all inflammatory and infective processes, such an assumption is practically equivalent to postulating an inflammatory lesion as the essential morbid process underlying disseminated sclerosis. Arguing on these lines the acute clinical disturbances are to be regarded as the result of an acute inflammatory process, and their partial or complete disappearance represents a dying down of the process with absorption of exudate and consequent functional recovery of the tissues; where however sufficient structural damage has occurred permanent effects will remain. The chronic progressive condition on the other hand, whether it supervenes on a previously more acute phase or whether it has characterized the illness *ab initio*, is evidence of the same morbid process, less intense in its action, but one nevertheless which never becomes entirely inactive. In such cases one expects to meet not so much new focal areas of disease in parts of the brain and cord some distance removed from those already affected, but rather a gradual extension of already existing areas and a consequent spread and intensification of already existing symptoms; in other words, the legs become more paraplegic, an eye more blind.

Again one cannot altogether ignore the existence of cases which after a variable time become definitely non-progressive, and we have already hinted at the possibility of the existence of spontaneous "cures." In the former cases, a small minority of the total, the disease has entirely ceased to be active, while the latter correspond to the "formes frustres" of many other infective processes.

The absence of cytological and chemical changes in the cerebrospinal fluid, as determined by ourselves in common with many other investigators, in no way militates against the hypothesis that the process is an inflammatory one. In the first place it seems probable that the increase in protein and morphological elements when occurring in other conditions should be ascribed to a diffuse or local reaction on the part of the meninges rather than to changes in the nervous parenchyma itself, and meningitic changes although present in some cases cannot be regarded as an invariable feature of the morbid histology of disseminated sclerosis. Secondly, pathological changes in the cerebrospinal fluid may be conspicuous by their absence in such frankly inflammatory conditions as acute poliomyelitis and encephalitis lethargica.

Histological investigations confirm the arguments based on clinical studies. Our own histological examinations have unfortunately been far from complete, but as far as they go they are in agreement with the more recent work which insists on an inflammatory reaction, acute or subacute, in the affected areas. Both Siemerling and Raecke, and Dawson describe a general tissue reaction in the early, and an essentially glia reaction in the later stages, the general architecture of the tissues being to a great extent preserved owing to the relative escape of axis cylinders and ganglion cells. Although the inflammatory phenomena of the earlier stages occupy a more prominent position in the description published by the German writers than in that given by Dawson, the latter nevertheless expresses the opinion that "there is overwhelming evidence that the great majority of the areas have arisen on an inflammatory basis." Both sets of observers lay stress on the fact that in the same individual different stages of the morbid process can be identified in different areas, some of recent origin and others of long standing, a state of affairs entirely in keeping with the clinical histories so characteristic of the remittent type of disseminated sclerosis. Moreover at the edges of areas even where sclerosis is complete there is histological evidence that the morbid process has never become entirely inactive, but is capable either of lighting up acutely, or, and perhaps more frequently, of advancing slowly by peripheral extension. In this connection we hesitate to accept the suggestion put forward by Siemerling and Raecke that the scar tissue formed by the glia proliferation protects the tissues from further damage. On the contrary we consider it much more probable that the virus lies latent in the scar tissue, but capable of initiating further infection when the local or

general conditions permit: in other words we regard the scar tissue as the starting-point from which reinfection originates. Finally both sets of observers agree that the histological changes can only be explained by a variation in the intensity of the morbid process in different areas; thus while rapid destruction of tissue is a feature in some, in others there is little more than a spreading gliosis, which they attribute to the leakage of a diluted virus from the essential primary lesions, and it is on this latter basis that the chronic progressive case finds its readiest explanation.

As regards the origin of the morbid process underlying disseminated sclerosis our own investigations have failed to throw any light on the subject; in no case have we succeeded in transmitting the disease to animals, while our bacteriological and parasitological investigations have been without result. Neither can we admit the claim of those writers, whose work we have reviewed, that they have transmitted the disease to rabbits and guinea-pigs. Even if we admit that in certain instances they have transmitted *a* disease, there is an entire absence of histological proof that the disease so transmitted was disseminated sclerosis. In view however of Steiner's monkey experiment it would seem advisable to continue experimental investigations with *Macacus rhesus*. Neither can we regard the finding of spirochætes in the hepatic vessels and ventricular fluid of inoculated guinea-pigs and rabbits as being of any decisive significance, in the absence of adequate evidence that the animals which harboured the parasite were suffering from disseminated sclerosis. Moreover it is not fully known to what extent spirochætes occur in animals under different conditions of health and disease; they have been found, for example, by one of us (L.S.D.) in the livers of apparently healthy guinea-pigs, while according to Fiessinger they are present in human urine in over 2 per cent. of normal individuals.

Adams in a recent contribution is inclined to support the spirochætal origin of the disease partly on clinical grounds, but mainly owing, first, to the presence in the cerebrospinal fluid of 95 per cent. of his cases of a luetic or paretic reaction to colloidal gold, secondly to the modifications which this reaction undergoes under neo-salvarsan treatment, and thirdly to the beneficial clinical effects of anti-syphilitic treatment in early cases of the disease. We feel however that until the rationale of the colloidal gold reaction is more completely understood, arguments based on the presence and behaviour of the reaction are unsubstantial, while in view of the remissions so characteristic of the disease the

beneficial effects claimed for any therapeutic measures must be treated with the greatest reserve.

Finally the finding of spirochætes in the cerebral hemispheres of two patients dead of disseminated sclerosis needs confirmation and amplification before we should be justified in accepting unreservedly the spirochætal origin of the disease.

While therefore we are of opinion that in spite of recent investigations the origin and nature of the morbid agent has yet to be discovered, we believe that there are sound reasons, both clinical and histological, for the view which regards the morbid process underlying disseminated sclerosis as inflammatory in character.

Conclusions.

(1) It is useful to recognize two clinical types of disseminated sclerosis: (*a*) the remittent type characterized by acute exacerbations at widely varying intervals alternating with quiescent periods, and (*b*) the chronic progressive type.

(2) In the present series (thirty-five patients) the proportion of remittent to chronic progressive cases is as six is to one.

(3) In early cases of the remittent type, once the acute disturbance has subsided, the patient may present no clinical evidence of organic disease over prolonged periods. The possibility of spontaneous cure cannot therefore be entirely denied.

(4) The remittent type in its later stages tends to assume the characteristics of the chronic progressive type.

(5) The great bulk of clinical and histological evidence is opposed to the view that these two types correspond to two different pathological processes. On the contrary, they are to be regarded as manifestations of one and the same disease, namely, disseminated sclerosis.

(6) Cultural and microscopic examination of the cerebrospinal fluid has in our hands thrown no light on the pathogenesis of the disease, and no specific organism has been isolated.

(7) Our attempts to transmit disseminated sclerosis from man to animals (rabbits) have been unsuccessful.

(8) We regard the transmissibility of the disease from man to animals as unproved.

(9) We are of opinion that the evidence in favour of the assumption that the pathogenic agent is a spirochæte is incomplete and in many

respects unsatisfactory, and we consider that the origin and nature of the morbid agent must for the present remain *sub judice*.

(10) We consider that the clinical and histological evidence is overwhelmingly in favour of the view that the morbid process underlying the disease is inflammatory in character.

APPENDIX.

Clinical Notes of Non-Fatal Cases.

Case III.—E. B., girl, aged 18, domestic servant. Admitted to National Hospital, Queen Square, on April 28, 1913.

Family and past history: Only child. Two sisters stillborn. Always delicate.

History of present illness: September, 1912: Retro-ocular pain with rapid failure of vision, so that she could not see her hand held in front of her eyes. October, 1912: Pain disappeared; vision improved so that she could see to get about; noticed difficulty in sitting up in bed unsupported. January, 1913: Tremor of left hand when she used it; could not hold a cup without spilling it; the condition cleared up within three weeks. February, 1913: Sudden weakness of right leg, unable to walk without support since this date; difficulty in holding her water, numerous "accidents"; this lasted for three weeks, and was then replaced by hesitancy of micturition which persists. March, 1913: Paræsthesiæ in right leg; never diplopia, dysarthria, or loss of emotional control.

On examination, April 28, 1913: Visceral examination negative. Wassermann reaction (blood) negative. Mental condition, normal. Vision: acuity, $\frac{1}{60}$, right and left. Both discs are pale all over, somewhat opaque, retinal pigment disturbed, edges clear (primary atrophy with probably a little antecedent œdema). Hearing, normal. Cranial nerves: pupils normal; congenital concomitant strabismus; no diplopia; nystagmus to right > left; weakness right lower face, otherwise negative. Motor.—Head and neck, natural. Upper extremities: no paresis; both arms a little unsteady, left > right; slight sensory ataxia on right; trunk muscles weak; no wasting; umbilical excursion upwards. Lower extremities: considerable weakness and spasticity, right much weaker than left; no wasting or obvious ataxy. Gait: spastic, reeling and very feeble; cannot walk unsupported. Sensory.—Subjective: numbness right leg; girdle sensation. Objective: slight astereognosis and raised threshold to compasses in right hand; diminution of tactile and painful sensibility on right from fifth rib to middle of thigh; some loss of sense of passive position and movement in ankles and toes, right > left; vibration much impaired in lower limbs. Sphincters: hesitancy of micturition. Reflexes: all jerks very brisk, lower > upper limbs; all abdominals absent; both plantars extensor.

Progress: The patient got gradually worse the whole time that she was under observation. July 21, 1913: Numerous paræsthesiæ in all limbs and trunk; both legs very spastic and almost completely paralysed; lower abdominal muscles are also spastic and paralysed; numerous flexor spasms in legs; sensory loss below fourth rib to touch and prick is increasing right > left; intention tremor has developed in both arms, left > right; profound asynergia and dysmetria; well sustained horizontal nystagmus; complete incontinence of urine and fæces; no bed-sores or pyrexia; vision *in statu quo*. December 8, 1913: Complete paraplegia in flexion: sensory loss to all forms below fourth rib is now profound, complete on right, almost so on left; general condition remains fairly good in spite of incontinence.

Summary: Sudden onset, remissions, partial recoveries, then steady and rather rapid progression downhill with few fresh symptoms.

Case IV.—N. C., woman, aged 32, widow. Admitted to National Hospital, Queen Square, on October 31, 1913.

Family history: Father dropped down dead, aged 43, from a ruptured aortic valve. Mother died of pulmonary tuberculosis. Several brothers and sisters alive and well.

Past history: Good. Husband died, aged 29, of rheumatic heart disease. One daughter died of diphtheria; one boy, aged four, alive and well. No miscarriages.

History of present illness: Four years ago sudden attacks of severe rotatory vertigo without deafness or tinnitus; these persisted for two years. Three years ago noticed curious fleeting sensations of numbness in legs and tightness round waist, which have never quite disappeared. Four months ago woke up one morning and found that her left hand was completely paralysed and she had lost all feeling below the left elbow. The right hand was numb and peculiar. She could not tell what she held in her hand unless she looked to see, and her writing became a scribble. She also had a tight band across the stomach and both legs were weak and quite numb; she could not feel hot or cold water in her legs, her walking was very unsteady "as if I was walking on air." The legs have improved, feeling has returned and she can walk better, but cannot even stand unsupported in the dark. The right hand has not altered, but the loss of sensation in the left upper limb gradually extended up to the shoulder, so that the whole arm "has an existence apart from me"; it is not paralysed. No affection of vision, speech, or sphincters.

October 31, 1913: Visceral examination negative; no developmental defects. Vertebral column healthy. Wassermann reaction (blood) negative. Mental condition: normal emotional tone; good type. Vision: acuity, $\frac{6}{12}$ left, $\frac{6}{9}$ right: fields normal; temporal pallor of both discs, left > right. Hearing normal. Cranial nerves: well sustained slow horizontal nystagmus, otherwise normal. Motor: head and neck natural, no rigidity. Upper extremities.—Right: hypotonia distally, no paresis with eyes open; no wasting or trophic changes; all finer movements of fingers characterized by overaction;

fingers wander with eyes shut; sensory ataxy. Left: profound hypotonia and complete inability to apply muscular power with eyes shut; no wasting. Trunk: slight diaphragmatic weakness; nothing else to note. Lower extremities: considerable weakness, left > right; no spasticity; no wasting or obvious ataxy. Gait: weak and ataxic; Rombergism present. Sensory: subjective—paræsthesiæ, limbs and trunk. On examination: complete loss to all forms below shoulder in left upper limb except algometer pressure which is recognized but not localized; loss of sense of passive position and movement is almost complete even at shoulder-joint; slight impairment of cutaneous sensibility in right hand below wrist; partial astereognosis right hand, compass threshold raised, and sense of passive position and movement impaired in wrist and fingers; to the left of the mid-line from the second cervical segment down to the foot there is profound loss to touch, pin-prick and temperature, and a similar condition prevails on the right below the umbilicus; there is profound loss to vibration in left pelvis and lower limb, and a slight impairment in right pelvis and lower limb; the sense of passive posture and movement is normal in the right lower limb, but slightly defective in the left. Sphincters natural. Reflexes: all tendon jerks brisk: all abdominals absent; both plantars indefinite, but probably extensor.

Progress: Two months later the sensory loss over the left arm was not quite so deep, and the patient had some idea of the position of the limb, but the ataxia with eyes shut was still very wild. The loss of deep sensation in both legs had increased and the gait was more ataxic. The right plantar response was definitely extensor, the left indefinite. Otherwise no change.

Summary: This case is regarded as doubtful, seeing that the condition can be explained by an extensive lesion in the upper cervical cord, such as one sees in syringomyelia. On the other hand the previous history of vertiginous attacks, paræsthesiæ, and the presence of temporal pallor points to a dissemination of lesions, and renders the diagnosis of disseminated sclerosis probable, although by no means certain. Attempts to trace this patient have not been successful.

Case V.—N. R., male, aged 32, farmer. Admitted to National Hospital, Queen Square, on November 3, 1913.

Family history: Nothing of importance.

Past history: When aged about 15 he injured the left shoulder (? fracture dislocation); his arm wasted above the elbow and never recovered. In 1904 he contracted a wart-like, venereal sore which was said to be not syphilitic: no evidence of syphilis ever developed.'

History of present illness: In 1899 he lost the sense of taste on the left side and his tongue and face felt dead and numb. This disappeared entirely in a few weeks. In 1906 he had a severe attack of furunculosis, and during convalescence he rather suddenly became aware of weakness of the left leg, his gait became unsteady, and he often vomited. His legs then became so weak that he could hardly get about, and he later developed diplopia and urgent micturition with frequent "accidents." After some months he began

to improve, the diplopia disappeared, and his legs recovered sufficiently to allow him to get back to his ranch and do his ordinary work. In 1909 he relapsed and could hardly walk unsupported; he was admitted to Queen Square and was found to have a typical spastic paraplegia, but no other physical signs beyond wasting of the muscles round the left shoulder. During the past four years his condition has varied considerably, there have been no new symptoms beyond weakness of the legs and loss of control over the bladder.

November 3, 1913: Good type and physique. Visceral examination negative: no evidence of syphilis. Wassermann reaction (blood) negative. Mental condition normal. Vision: acuity, $\frac{6}{24}$ right, $\frac{6}{12}$ left; temporal pallor of both discs. Cranial nerves: horizontal nystagmus to right and left, otherwise negative. Motor.—Head and neck normal; upper limbs normal, except for atrophic palsy of muscles round left shoulder and of upper arm, due to old injury; no tremor or ataxia. Lower limbs: severe spastic paraplegia; very little power at any joint, numerous flexor spasms. Sensory.—Subjective: *nil*. Objective: blunting of tactile sensibility in right leg; some loss of sense of passive position and movement in ankles and toes; vibration not tested. Sphincters: retention of urine; requires catheter. Gait: impossible. Reflexes: all jerks greatly increased; all abdominals absent; both plantar responses extensor.

Progress: Condition unchanged two months later.

Summary: Remittent type in early stages; in later stages assuming a chronic progressive course with absence of new symptoms.

Case VI.—P. E., male, aged 27. Admitted to National Hospital, Queen Square, on August 15, 1913.

Family and past history: Nothing of importance.

History of present illness: Seven years ago he developed diplopia, which persisted for six months. Soon after he had begun to see double he experienced difficulty in holding his water, and walked as if drunk. His walking gradually deteriorated, and he lost all control over his bladder, but after several months both these symptoms improved. The improvement, however, was not maintained, his walking became more unsteady than ever, and his hands began to shake so that he had difficulty in feeding himself. Later his head shook when he talked and his speech became affected. During the past five years he has got gradually worse, but no fresh symptoms have developed. Never paræsthesiæ or vertigo.

On examination, August 15, 1913: Fair type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition: Unduly sanguine; defective emotional control. Vision: acuity, $\frac{6}{36}$ right and left (uncorrected); well-marked temporal pallor of both discs. Hearing normal. Cranial nerves: jelly-like nystagmus with eyes at rest; coarse horizontal nystagmus on looking to right and left; weakness of left external rectus and diplopia on looking to left; typical scanning, staccato

speech, very tremulous, with explosive element well marked. Motor: head and neck, nodding tremor when unsupported, greatly accentuated when he talks; upper extremities no paresis, typical intention tremor right and left, dysmetria and asynergia readily demonstrable; trunk normal, can sit up without use of hands; lower extremities, tone increased, slight weakness of dorsi-flexors at ankles, tremulous. Sensory: subjective and objective, nothing to note: vibration not tested. Sphincters: urgent micturition: frequent "accidents." Gait: reeling and drunken, characteristic of cerebellar inco-ordination; steps unequal in size and direction; no Rombergism. Reflexes: all jerks greatly increased; all abdominals absent; both plantar responses extensor.

Progress: Four months later *in statu quo.*

Note: Chronic progressive type; prominence of cerebellar signs and symptoms.

Case VII.—W. S., male, aged 21. Admitted to National Hospital, Queen Square, on October 31, 1913.

Family and past history: Nothing to note.

History of present illness: Three years ago his arms began to shake when he used them and his speech altered; a little later he walked as if drunk. The general unsteadiness of all four limbs and the affection of speech have since got progressively worse, and he laughs for hours together for no apparent reason. He has been absolutely helpless for twelve months, and more recently has lost control over his bladder. Never ocular disturbance or paræsthesiæ.

On examination, October 31, 1913: Poor type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition: intelligence below the average; silly and giggling; laughs when spoken to and at his own tremor. Vision: acuity, $\frac{6}{9}$ right and left; temporal pallor of both discs. Cranial nerves: coarse nystagmus in all directions, no ophthalmoplegia; speech typical of advanced disseminated sclerosis, slow, staccato, explosive, and barely intelligible; some difficulty in swallowing, but never nasal regurgitation. Motor: no obvious paresis or tone changes; even when lying he is a mass of oscillations; tremor of head and limbs of extreme degree greatly accentuated by effort; unable to do anything for himself; dysmetria of upper limbs is well shown. Sensory: subjective and objective negative; vibration not tested. Sphincters: incontinence of urine, feels "call" and passage. Gait: impossible without support, attempts show wild cerebellar inco-ordination. Reflexes: all tendon jerks greatly increased; all abdominals absent; both plantar responses extensor.

Progress: One month later, *in statu quo.*

Note: Chronic progressive type; profound cerebellar inco-ordination of speech and limbs.

Case VIII.—V. B., male, aged 34, shop-fitter. Admitted to National Hospital, Queen Square, on October 3, 1913.

Family and past history good.

History of present illness: Ten years ago the right arm suddenly became useless: it was not paralysed but he could not control it properly nor distinguish what he held in his hand without looking. In three months the arm was perfectly well again. A little later he became unduly emotional, laughing and crying for no reason; this has got much more obvious lately. A year and a half ago his walking rather rapidly became affected and his friends thought he was drunk (patient is a teetotaller); this symptom has varied considerably. Latterly his left foot has dragged and he tires easily. At the same time he noticed curious sensations of "tightness" and numbness in his face and legs, followed by itching of the thighs; these lasted for six months. Recently he has had aching pain in both legs. Six months ago he experienced difficulty in holding his water; recently he has had difficulty in passing it, and has become impotent. He also noticed an alteration in his speech which became slow and awkward; this defect amuses him and he can always keep up his spirits by talking to himself. Four months ago his hands began to shake; his writing became bad and it took him half an hour to do his collar up. Two months ago his vision got rather poor, but has since improved. Never diplopia or vertigo. During the past eighteen months his condition has varied considerably and at times he seems almost well.

On examination, October 3, 1913: Good type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition: very intelligent; grossly defective emotional control; he laughs till he cries. Vision: acuity, $\frac{6}{9}$ partly, right and left; temporal pallor with fluffiness of inner edges of both discs, suggestive of preceding œdema. Cranial nerves: horizontal nystagmus on looking to right and left; no ophthalmoplegia; coarse tremor of tongue; typical dysarthria, staccato, jerky, explosive. Motor.—Head and neck: coarse nodding tremor when sitting and standing; no loss of power in limbs or trunk, no spasticity; well-marked intention tremor especially of arms, right > left: dysmetria and asynergia of both arms. Sensory.—Subjective: pains in legs. Objective: superficial sensation good; some impairment of sense of passive posture and movement in toes and ankles; vibration greatly impaired in pelvis and femora, lost below knees. Sphincters: hesitancy of micturition. Gait: slight dragging of feet: reeling and staggering, well marked disturbance of equilibration; no Rombergism. Reflexes: all tendon jerks greatly increased: all abdominals absent; both plantar responses extensor.

Progress: Two months later *in statu quo*.

Note: Remittent type of ten years' duration; early symptoms separated from later ones by a long interval (eight years). Cerebellar manifestations a prominent feature.

Case IX.—L. K., woman, aged 39, widow. Admitted to National Hospital, Queen Square, on November 25, 1913.

Family and past history negative. Two children, no miscarriages.

History of present illness: Two years ago began to have attacks of severe rotatory vertigo with headache and tinnitus in both ears; these gradually

disappeared and she has had none for over a year. Eighteen months ago she had severe retro-ocular pain and her vision rather rapidly failed, so that three months later she was only able to get about in familiar places. The condition has since remained stationary. One year ago she began to have trouble with the bladder, hesitancy of micturition alternating with precipitancy; occasional "accidents." For the past six months she has noticed curious sensations in arms and legs, "tightness," numbness, "tickling," &c. Never dysarthria, tremor, or disturbance of gait."

On examination, November 25, 1913: Normal appearance. X-ray of sella turcica normal. Sugar tolerance normal. Wassermann reaction (blood) negative. Visceral examination negative. Mental condition normal. Vision: counts fingers correctly at 3 ft., right and left; large scotoma right and left eye, extending into temporal more than into nasal field; both discs very pale—primary optic atrophy. Hearing normal. Cranial nerves: negative; no nystagmus. Motor: normal in every particular. Sensory.—Subjective: numerous paræsthesiæ. Objective: normal; vibration not tested. Sphincters: urgent micturition. Gait: natural. Reflexes: all jerks rather exaggerated; left abdominals normal, right much diminished; both plantar responses flexor.

Progress: One month later *in statu quo*.

Note: Atypical case, but clear evidence of dissemination of lesions. Severe bilateral visual failure.

Case X.—T. R., woman, aged 27, single. Admitted to National Hospital, Queen Square, on November 2, 1913.

Family history: Negative; one of a large family.

Past history: Trivial accident to right knee eighteen months ago, followed by painful swelling of joint.

History of present illness: One year ago the right leg became weak and dragged, and she has not walked about for six months. Weakness of the left leg noticed quite recently. (N.B.—Patient is a very unreliable witness and can give no accurate account of her illness.)

On examination, November 2, 1913: Poor type and physique. Visceral examination and vertebral column normal. Wassermann reaction (blood) negative. Mental condition: poor intelligence; facile, fatuous and inattentive; gross loss of emotional control; laughs at her own disabilities. Vision and hearing normal. Cranial nerves normal; no nystagmus or dysarthria. Motor.—Head and neck: natural. Upper limbs: no loss of power; both hands rather unsteady and tremulous; no very definite ataxia. Trunk: all abdominal muscles spastic and weak. Lower limbs: tone increased; considerable weakness, especially of flexors; numerous flexor involuntary spasms; spastic paraplegia. Sensory.—Subjective: normal. Objective: difficult to test, as examination amuses patient, rendering her an impossible witness; some impairment of both deep and superficial sensation below knees. Sphincters: urgent micturition. Gait: spastic, right leg > left. Reflexes:

all jerks very brisk, especially in lower limbs; all abdominals absent; both plantar responses extensor.

Progress: Two months later unchanged.

Note: Chronic progressive type; lesions not numerous.

Case XI.—F. R., male, aged 28. Admitted to National Hospital, Queen Square, on September 22, 1913.

Family and past history: Negative.

History of present illness: Two years ago his gait became unsteady and his right foot dragged. The condition has varied, but recently has become worse, so that he is unable to walk without support. One year ago he began to have difficulty in voiding his urine, but this has now disappeared. His arms became shaky and weak, especially his right, and he had difficulty in writing. Six months ago the vision in his left eye rather rapidly failed, but has improved recently. During the past three weeks he has been subject to attacks of severe giddiness accompanied by vomiting. Never dysarthria or diplopia; no paræsthesiæ.

On examination, September 22, 1913: Good type, fair physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition natural. Vision: acuity, $\frac{6}{6}$ right, $\frac{6}{24}$ left; no note of ophthalmoscopic examination. Hearing normal. Cranial nerves normal; no nystagmus or dysarthria. Motor.—Head and neck normal. Upper limbs: no spasticity; power below normal, right > left; moderate intention tremor with dysarthria and asynergia, right > left. Trunk normal. Lower limbs: spastic paraplegia in extension; right leg weaker than left; power in both greatly diminished. Sensory: subjective and objective normal; vibration not tested. Sphincters natural. Gait: impossible without support; very spastic. Reflexes: all jerks greatly increased, especially in legs; absent abdominals; both plantar responses extensor.

Progress: Three months later the condition had vastly improved; the intention tremor of the arms had largely disappeared and he could walk unaided. There was less spasticity, and the gait was more reeling and like that seen in cerebellar disease.

Note: Remittent type; onset of different symptoms never very acute.

Case XII.—E. T., woman, aged 35, married. Admitted to National Hospital, Queen Square, on December 11, 1913.

Family history: Nothing to note.

Past history: Has always been "nervous"; two children alive and well; no miscarriages.

History of present illness: Four years ago she complained of pains in the legs, her walking became unsteady, and her hands shook at times. She also became unduly emotional, crying and laughing for no reason, and suffered from urgent micturition with occasional "accidents." She remained in much the same condition for three years, varying round a more or less constant mean. Following a mental shock a year ago her walking became progressively

worse and her hands shook when excited. She now complains of aching pains in hips and arms, and her feet feel stone cold. The emotionalism has almost disappeared. Never ocular symptoms or dysarthria.

On examination, December 11, 1913: Good type, fair physique. Visceral examination, negative. Wassermann reaction (blood), negative. Mental condition, normal: reserved, and good emotional tone. Vision and hearing: normal in every particular. Cranial nerves: nothing to note except ill-sustained nystagmus. Motor.—Head and neck normal. Upper limbs: no loss of power; both hands are rather unsteady and awkward, but there is no true intention tremor or dysmetria. Trunk: very weak; cannot sit up without use of hands; abdominal muscles spastic. Lower limbs: spastic in extension; gross loss of power, left > right; no wasting. Sensory.—Subjective: paræsthesiæ in limbs, especially legs. Objective: slight loss to light touch and pin prick below knees, left > right; definite impairment of sense of position and movement at and below knees, right > left; vibration impaired slightly below eighth thoracic spine, and barely recognized in pelvis and lower extremities, especially on right. Sphincters: urgency of both: feels "call" and passage. Gait: very spastic; can walk a few steps without support; Rombergism present. Reflexes: all jerks exaggerated, lower > upper limbs; all abdominals absent; both plantar responses extensor.

Progress: Condition unchanged two months later.

Note: Chronic progressive type without exacerbations.

Case XIII.—J. B., male, aged 45, shop assistant. Admitted to National Hospital, Queen Square, on December 16, 1913.

Family history negative.

Past history: Always had good health; gonorrhœa twenty years ago; denies syphilis.

History of present illness: Six years ago his right foot began to drag; this got worse for a year and has remained stationary since. Four years ago he rather suddenly lost the use of his left hand; he could move his fingers, but they seemed clumsy, and he could not identify objects in his left trousers pocket. About the same time he had some difficulty in passing his water, which entirely disappeared after a few months. For the past three years he has been unnaturally nervous and apt to burst into tears, "a matter beyond my comprehension." One year ago his left leg began to get weak; his left arm also became rapidly much worse, and he now loses it in bed and it gets "out of control." Also, "When I move my right hand up to my mouth, the left arm goes across and catches hold of it." His left arm often feels numb and his feet cold. He has never noticed any loss of sensation in arms or legs. No defect of speech or vision, and no tremor.

On examination, December 16, 1913: Good type and physique, rather stout. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition normal; no loss of emotional control detected. Cranial nerves normal; no ophthalmoplegia or nystagmus. Vision and hearing

perfect. Motor.—Head and neck: no tremor, rigidity or wasting. Upper limbs: power at shoulder and elbow is impaired on left, but there is no sign of wasting and the electrical reactions are normal; power in hand is normal, there is a profound degree of sensory ataxia in the left arm and tabetic athetosis; in the right arm power is normal, except for flexors of wrists and fingers, which are definitely weak and wasted with lowered faradic excitability; no ataxia or tremor. Trunk good. Lower limbs: both spastic and weak, right much more so than left; flexors weaker than extensors; no wasting. Vertebral column normal. Sensory.—Subjective: occasional tingling in both hands. Objective: complete loss to light touch over left cervical segments 2 to 5 and slight impairment over left C. 6 and 7, and right C. 2 to 5; pin-prick and temperature lost or impaired over C. 2 to 7 on left, and C. 2 to 6 on right; profound loss of sense of passive position movement whole of left arm, complete in hand; no threshold for compasses in left hand, which is completely astereognostic; threshold to compasses in right hand = 2·5 cm.; no astereognosis; vibration lost in left upper limb, good in right; completely lost in spinal column below sixth spine, and impaired in left pelvis and lower limb. Sphincters natural. Gait: spastic, right leg > left; no ataxy or Rombergism. Reflexes: triceps jerks brisk, right and left; biceps and supinator present and normal on right, absent on left; all abdominals absent; both plantar responses extensor: associated movements are present in the upper limbs—e.g., grasping with right hand is associated with a similar movement in left.

Progress: No change three months later.

Note: This must be regarded as a very doubtful case. The patient insisted on his loss of emotional control, but it was not demonstrable during the time that he was under observation. The rest of the condition can be explained by a central lesion of the upper cervical cord such as occurs in syringomyelia; at the same time neither the clinical course nor physical signs can be regarded as typical of syringomyelia.

Case XIV.—M. B., woman, aged 31, married. Admitted to St. Thomas's Hospital on January 3, 1914.

Family history negative.

Past history: Chlorosis at age of 15. Three children alive and well; one twin stillborn (difficult labour); no miscarriages.

History of present illness: Eleven years ago she noticed pains and various odd sensations in her limbs, especially the left arm and leg, and simultaneously her walking became unsteady. A year later she became subject to sudden attacks of rotatory vertigo, in which she invariably fell to the left, accompanied by headache and noises in the ears; she has had none of these attacks during the past two years. Ten years ago she began to have difficulty in holding her water; this has gradually increased, and she now has frequent "accidents." Eight years ago the paræsthesiæ and difficulty in walking almost entirely disappeared, but after nine or ten months her walking rapidly got bad again, so that her friends thought she was drunk. At the same time she

became unduly emotional and "laughed at everything." These various symptoms have persisted, but vary enormously in their intensity from time to time, the improvements sometimes lasting for several months. On the whole, however, her legs have got weaker, and for six months she has been unable to walk at all. Six months ago her left hand began to shake, and she had difficulty in using a fork. Two months ago she was told that her speech was "funny," but she never noticed any alteration herself. Three weeks ago she woke up with a squint, and has seen double ever since.

On examination, January 3, 1914: Good type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition: very intelligent; defective emotional control. Vision: acuity, $\frac{6}{12}$ right, $\frac{6}{9}$ left; both discs pale all over and clear cut. Hearing normal. Cranial nerves: weakness right external rectus with internal strabismus; fine nystagmus of moderate speed with both lateral and vertical movements; no dysarthria. Motor.—Head and neck normal. Upper limbs: right normal; slight weakness of hemiplegic type on left, with intention tremor and dysmetria. Trunk normal. Lower limbs: spastic paraplegia in extension; flexors much weaker than extensors and left much weaker than right. Sensory.—Subjective: numerous paræsthesiæ. Objective: only loss discovered is to vibration, which is lost or impaired in pelvis and lower limbs; left > right. Sphincters: urgent micturition; feels "call" and passage. Gait: spastic and ataxic; well marked disequilibration and asynergia; no Rombergism. Reflexes: all tendon jerks much exaggerated: abdominals absent; both plantar responses extensor.

Progress: Six weeks later the gait was much improved and she could walk a few steps unaided.

Note: Remittent type with exacerbations and wide dissemination of lesions.

Case XV.—H. S., woman, aged 25. Admitted to St. Thomas's Hospital on January 1, 1914.

Family history negative.

Past history: Asthma up to the age of 16.

History of present illness: Eighteen months ago woke up to find that she saw double, and that she could not shut the right eye, which was turned outwards. In six months time she was quite well again. Six months ago her walking rather rapidly became unsteady, and she reeled about as if drunk. One month ago her walking became much worse, the diplopia recurred, and the left arm suddenly became useless, so that she could not control it or feel what she held in her hand. She also noticed numbness of the right leg and aching pain in the right arm and left leg. Never dysarthria or sphincter disturbance.

On examination, January 1, 1914: Good type, poor physique. Visceral examination: congenital pulmonary stenosis; no cyanosis; otherwise negative. Wassermann reaction (blood) negative. Mental condition: intelligent;

poor emotional tone; unduly sanguine. Vision: acuity, $\frac{6}{24}$ (corrected), right and left; central scotoma for colour in both eyes; discs distinctly pale, especially in temporal halves. Hearing: normal. Cranial nerves: weakness of right internal rectus; rapid nystagmus on looking to the right, otherwise negative; no dysarthria. Motor.—Head and neck: nodding tremor when unsupported. Upper limbs: good power; no intention tremor; profound sensory ataxia on left with tabetic athetosis. Trunk normal. Lower limbs: slight spasticity with some weakness of flexors. Sensory.—Subjective: paræsthesiæ in limbs. Objective: superficial sensation perfect: profound loss of sense of passive position and movement in left arm, complete in fingers; complete astereognosis, no threshold obtainable to compasses; vibration not tested. Sphincters natural. Gait: no obvious spasticity, but profound cerebellar inco-ordination; no Rombergism; cannot walk without support. Reflexes: all jerks increased; all abdominals absent; both plantar responses extensor.

Progress: Ten weeks later the walking had improved, but was still very inco-ordinated. Otherwise *in statu quo*.

Note: Typical remittent type with wide dissemination of lesions.

Case XVI.—E. M., woman, aged 25, married. Admitted to National Hospital, Queen Square, on October 23, 1913.

Family history: Mother died of "creeping paralysis," aged 47.

Past history: Good. Three healthy children, no miscarriages.

History of present illness: Six years ago, three months after a normal confinement, her walking became unsteady, her left hand weak, and she had difficulty in passing her water. Nine months later all these symptoms became greatly aggravated, micturition became urgent, she developed diplopia, her sight began to fail, she lost control over her emotions, and had frequent boring pains in the legs. At this time she was admitted to the National Hospital, when the note was as follows: Visual acuity, $\frac{6}{24}$ right and left, temporal pallor, nystagmus, intention tremor of arms, spastic paraplegia, with increased tendon reflexes, absent abdominals, and double extensor plantar responses. She rapidly improved, the diplopia disappeared, and except for some slight unsteadiness on her legs she remained quite well for four years. Two years ago, three months after her third confinement, her walking rapidly deteriorated again, and she can now only get about the house by holding on to things. Six months ago micturition again became urgent, but there have been no "accidents." Two months ago she suddenly experienced difficulty in getting her words out and her head began to shake when she talked. Her right hand had also become shaky; and her legs go quite numb from the knees downwards.

On examination, October 23, 1913: Good type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition: rather sanguine; no loss of emotional control. Vision: acuity, $\frac{6}{12}$ right, $\frac{6}{24}$ left: right disc generally greyish but whiter in temporal half, edges

clear; left disc very white and atrophic looking. Hearing normal. Cranial nerves: no ophthalmoplegia; jelly-like nystagmus with eyes at rest: quick nystagmus of wide amplitude on looking in all directions. Articulation slow, scanning, syllabic, and explosive; slight tremor of tongue. Motor.—Head and neck: nodding tremor when unsupported. Upper limbs: no weakness; intention tremor of moderate degree in both arms; well-marked dysmetria, right and left. Trunk: rather weak; no local palsy. Lower limbs: both a little spastic, considerable weakness, right > left, especially of flexors, and well marked intention tremor. Sensory.—Subjective: paræsthesiæ in legs. Objective: no disturbance except slight impairment of sense of position in ankles and toes; vibration not tested. Sphincters: urgent micturition. Gait: only possible with support; generalized tremor, reeling and drunken, slight spasticity; no Rombergism. Reflexes: all jerks greatly increased; all abdominals absent; both plantar responses extensor.

Progress: Unchanged two months later.

Note: Typical remittent case; onset and exacerbation related to confinement. Generalized cerebellar ataxia.

Case XVII.—L. E., woman, aged 34. Admitted to St. Thomas's Hospital on February 12, 1914.

Family history negative.

Past history: Rheumatic fever, aged 15. Always been "hysterical."

History of present illness: Two years ago her left arm and leg suddenly became weak; recovery within a week. Two months later she noticed that she was blind in the left eye, and began to experience tightness and tingling in the right arm and hand, which has persisted. Six months ago her walking became unsteady, especially in the dark. No dysarthria, diplopia, or sphincter trouble.

On examination, February 12, 1914: Poor type, good physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition: highly suggestible; good emotional tone. Vision: acuity, $\frac{6}{24}$ left, $\frac{6}{9}$ partly right; concentric limitation of visual fields; left disc pale all over, edges clear; right disc slight temporal pallor. Hearing normal. Cranial nerves negative, no nystagmus. Motor: No paresis or intention tremor; definite sensory ataxia of right arm with well-marked tabetic athetosis. Sensory: various disturbances of cutaneous sensation can be manufactured or dispersed by suggestion; compasses threshold 1·0 cm. left palm, 3·0 to 3·5 cm. right palm: astereognosis right hand and disturbance of sense of passive posture and movement; questionable impairment of deep sensation in right leg; vibration not tested. Sphincters natural. Gait: slight tabetic ataxia of right leg; Rombergism present. Reflexes: all tendon jerks increased, right > left; all abdominals absent: right plantar response extensor, left flexor.

Progress: The patient was readmitted three months later with an hysterical gait, which disappeared under treatment.

Note: Remittent type with superadded hysterical manifestations.

Case XVIII.—E. C., woman, aged 33. Admitted to National Hospital, Queen Square, on February 19, 1914.

Family and past history: Good. Mild influenza two months ago.

History of present illness: Three weeks ago the left hand suddenly "went funny and useless" so that she could not feel things properly, followed two days later by weakness of the left leg and urgent micturition. She had a severe headache which persisted for four days, and then gradually disappeared. No vomiting, vertigo, or diplopia. The left hand now feels numb and cold.

On examination, February 19, 1914: Good type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition, vision and hearing normal. Cranial nerves: no nystagmus; definite weakness of right lower face with emotional movements only; otherwise negative. Motor.—Head and neck normal. Upper limbs: right normal; no paresis of left arm except when eyes are closed or limb is screened from view during testing; tabetic athetosis of left hand with pronounced sensory ataxy of whole limb. Trunk normal. Lower limbs: extended and adducted with some extensor rigidity, left much > right; some weakness especially of flexors on left, power good on right. Sensory: slight impairment to touch and pin-prick below left elbow; partial astereognosis of left hand, compass threshold 4·5 cm. left palm; impaired sense of passive posture and movement whole of left upper limb, more marked distally; vibration not tested. Sphincters: urgent micturition. Gait: spastic, left much greater than right; no gross inco-ordination; Rombergism negative. Reflexes: all tendon jerks exaggerated, left > right; abdominals diminished on left, normal on right; both plantar responses extensor.

Progress: The patient rapidly improved. One month later the right plantar response was flexor, sphincter control was normal, and the loss of deep sensibility in the left arm was much less.

Note: Early case; acute onset with lesions in spinal cord and subthalamic region, and rapid improvement.

Case XIX.—F. G., woman, aged 23. Admitted to St. Thomas's Hospital on June 8, 1914.

Family history good.

Past history: Always high spirited. Six years ago had "St. Vitus's dance," and "could not control right arm"; well in six weeks. Otherwise good.

History of present illness: One month ago she experienced pains in both hands and forearms, especially the left, with numbness and loss of feeling. This got worse for four days until the hands were "useless"; they were not paralysed, but she could not feel what she held in her hands and dropped objects without being aware of it. The numbness has now spread up beyond the left elbow. At the same time her legs felt numb, and she felt as if a belt were tied round her chest; both these symptoms rapidly disappeared. One week ago she had a sensation as if someone were pouring water down her

neck and it was trickling down her left side, and she began to have difficulty in holding her water, but has had no "accidents." No other symptoms; walking entirely unaffected.

On examination, June 8, 1914: Good type: physique much above average. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition, vision and hearing normal; first-rate witness. Cranial nerves: no ophthalmoplegia; slow nystagmus of moderate amplitude on conjugate deviation to the right; otherwise negative. Motor: Head and neck normal. Upper limbs: there is no gross paresis, but on the left there is a slight but appreciable weakness of the biceps and flexors of wrist and fingers; tested with the eyes shut there is an apparent weakness of all muscles: no wasting or tremor; the movements of both hands, the left more than the right, are extremely awkward and suggestive of spasm, yet on manipulation no spasm is present; there is well-marked sensory ataxia (left > right) with loss of direction, and with the arms outstretched and eyes closed the fingers and hands wander into all kinds of grotesque positions of which the patient is unconscious; writing is only possible with the eyes open. Trunk and lower limbs: normal in every particular. Sensory.—Subjective: numbness of both hands. Objective: slight impairment of tactile sensibility in left arm and hand, corresponding roughly to C. 5 and 6 segments; prick, temperature and localization perfect; profound loss to compasses both hands, threshold 5 cm. right, 7·5 cm. left; vibration slightly diminished left hand and carpus, normal elsewhere; passive position and movement—profound loss distally in both upper limbs (left > right), good at shoulders; complete loss to size, shape, weight and consistence in left hand, and almost complete loss in right. No disturbance in trunk or lower extremities. Sphincters: precipitate micturition. Gait normal. Reflexes: all tendon reflexes active except left biceps jerk which is absent; abdominals present and equal, both plantar responses flexor.

Progress: Three weeks later the left biceps jerk had returned, sphincter control was normal, there was no appreciable disturbance of deep sensation in the right hand, and that in the left was much less intense. Nystagmus was still present. Six months later (January, 1915) the patient felt perfectly well, and the only physical sign was nystagmus on looking to the right. In January, 1920, the patient was seen again; she had remained in perfect health, was married with two healthy children, and there was no physical sign of organic disease.

Note: Sudden onset with symptoms pointing to lesion of posterior columns in upper cervical region. Rapid recovery and disappearance of all physical signs; in perfect health six years later.

Case XX.—R. I., girl, aged 19. Admitted to St. Thomas's Hospital on June 2, 1914.

Family history: Twin sister excitable and emotional.

Past history: Excellent.

History of present illness: One year ago she was noticed to be unduly irritable and peevish, and complained of her legs feeling tired; shortly afterwards her walking become unsteady and she fell down if she attempted to run. Ever since she has staggered as if drunk and now can barely walk without support. At the same time she began to have attacks of uncontrollable giggling and laughing, and had difficulty in holding her water, with numerous "accidents." She also complained of curious sensations in her legs, which felt numb and "too full." Her hands also became unsteady, especially the left, so that she could not play the piano and her writing deteriorated. No alteration of speech, but her singing master noticed that her singing voice was shaky. No ocular symptoms or vertigo. The condition has slowly got worse, but no fresh symptoms have appeared.

On examination, June 2, 1914: Good type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition: intelligent and observant; very sanguine and indifferent as regards her disabilities; defective emotional control. Vision and hearing, normal. Cranial nerves: inconstant nystagmus on conjugate deviation, slow jerks on looking to the right, rapid to the left; otherwise negative; no dysarthria. Motor.—Head and neck, normal. Upper limbs: no loss of power; moderate intention tremor, left much > right, with asynergia and dysmetria. Trunk: No weakness; combined flexion of hip and trunk well shown. Lower limbs: adducted and extended; considerable extensor spasm; weakness left > right, mainly of flexors at all joints and abductors at hip-joints; intention tremor present. Sensory.—Subjective: numbness of legs. Objective: cutaneous sensibility perfect; impairment to compasses and passive position and movement left lower limb; complete loss to vibration in lower limbs, profound loss in pelvis and lumbo-sacral spine, impaired in lower thoracic spine. Sphincters: urgent micturition. Gait: slighly spastic, left > right; Rombergism doubtful; general disequilibration, tottering and staggering; no synergic movement of arms; cannot bend back without losing balance. Reflexes: tendon jerks in arms exaggerated, in legs multiple: all abdominals absent: both plantar responses extensor.

Progress: Two months later impairment of deep sensation in lower limbs had increased, Rombergism was definitely present, and walking was impossible without support. Nystagmus was constantly present.

Note: Chronic progressive type with wide diffusion of lesions in brain, brain-stem and cord.

Case XXI.—I. W., woman, aged 25. Admitted to St. Thomas's Hospital on August 27, 1914.

Family and past history good.

History of present illness: Six years ago she suddenly became blind in the left eye; after a week the sight rapidly returned. Just over a year ago she had a "nervous breakdown," the chief symptoms being severe aching pains in the legs and back, lassitude and emotional instability; she has never been well since. Five months ago she was drenched in a thunderstorm and caught a

chill; her legs felt numb and painful and she had pins and needles in her fingers. After a few days in bed she got up to find that her walking was unsteady, and this has got worse; she reels about like a drunken person and is at times unable to walk without assistance. Quite recently she has had attacks of rotatory vertigo. No diplopia, dysarthria, or sphincter trouble.

On examination, August 27, 1914: Good type and physique. Visceral examination, negative. Wassermann reaction (blood) negative. Mental condition: unduly optimistic and sanguine; defective emotional control. Vision and hearing normal; visual acuity is $\frac{6}{6}$ in both eyes, and both optic discs are of good colour. Cranial nerves: nystagmus on lateral movements, slow to the left, rapid to the right; otherwise negative. Motor.—Head and neck: nodding tremor when unsupported. Upper limbs: no paresis; fine static tremor. Trunk: good. Lower limbs: no definite paresis; slight extensor spasticity. Sensory.—Subjective: paræsthesiæ in limbs, legs > arms. Objective, normal. Sphincters natural. Gait: very ataxic, reels about; profound asynergia, typical of cerebellar disease; no Rombergism. Reflexes: All jerks very brisk; all abdominals absent; both plantar responses extensor.

Progress: No change two months later. Six months later patient was reported to have developed difficulty in talking and severe tremor of both arms, but these findings have not been included in the statistical table.

Note: Remittent type; no objective evidence of optic nerve lesion in spite of history of sudden amaurosis six years previously.

Case XXII.—G. R., male, aged 22. Admitted to St. Thomas's Hospital, January 19, 1915.

Family and past history, good.

History of present illness: Five years ago his speech began to drawl and he experienced tight feelings round his knees. Later he began "to get shaky all over," especially when excited, and he "got in a tangle with his words." His walking became unsteady as if he were drunk. In July, 1911, he was admitted to St. Thomas's Hospital, when the following note was made: Scanning speech, nystagmus, slight intention tremor of arms, cerebellar gait, exaggerated reflexes, double extensor plantar response. On discharge two months later he had much improved. He remained fairly well for three years, experienced no more paræsthesiæ, and his walking at times seemed almost normal. Some months ago he noticed the vision of his left eye to be failing, and six weeks ago all his symptoms gradually got much worse again. He became very emotional and excitable, his speech at times was barely intelligible, and his walking more unsteady than ever. He also got very sleepy and seemed unnaturally hungry. No vertigo, diplopia, or sphincter trouble.

On examination, January 19, 1915: Good type and physique. Visceral examination, negative. Wassermann reaction (blood) negative. Mental condition: excitable; laughs at his own tremor. Vision: acuity, $\frac{6}{18}$ left, $\frac{6}{9}$ right; scotoma to colours in left eye in nasal field, not quite reaching fixation point; temporal pallor of both discs, left > right. Hearing, normal. Cranial

nerves: horizontal nystagmus; weakness of right lower face, especially with emotional movements. Typical dysmetria, slow syllabic, tremulous, monotonous, with overaction. Motor.—Head and neck: to-and-fro tremor, accentuated when he talks. Upper limbs: no paresis; slight intention tremor, especially with rapid movements; dysmetria and dysdiadokokinesis well marked; breaks pencil when writing. Trunk: combined flexion of hip and trunk; some tremor in sitting position. Lower limbs: no paresis; tremulous and awkward. Sensory: subjective and objective, normal. Sphincters, natural. Gait: staggering and reeling; gross asynergia; no Rombergism. Reflexes: all tendon jerks exaggerated; abdominals brisk and equal; right plantar response extensor, left indefinite.

Progress: Unchanged four weeks later.

Note: Remittent type with little tendency for development of fresh lesions. Cerebellar inco-ordination a prominent feature.

Case XXIII.—E. C., woman, aged 27, nurse. Admitted to St. Thomas's Hospital on February 1, 1915.

Family history: Father paralysed in legs for thirty years.

Past history good.

History of present illness: Seven months ago suddenly developed diplopia, which persisted for four days. A month later she suddenly lost the use of her left leg and fell down in the street; the leg seemed absolutely numb. Two weeks later the power and sensation began to return, and in another two weeks her walking was almost normal again. About this time she began to notice curious sensations in both legs and round her waist, "tight feelings," cramps, tinglings, &c. Three months ago she experienced urgency of micturition and the left leg again suddenly became weak. No tremor, dysarthria, or vertigo.

On examination, February 1, 1915: Good type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition normal; first-class witness. Vision and hearing normal, except for a very doubtful temporal pallor of both discs. Cranial nerves: slow nystagmus on looking to left, otherwise negative. Motor.—Head and neck, trunk and limbs, normal. Lower limbs: slight extensor spasticity with weakness of flexors on left, power good on right, no ataxia. Sensory.—Subjective, paræsthesiæ. Objective: normal in every particular. Sphincters: slight urgency of micturition. Gait: weak and spastic, no Rombergism. Reflexes: all tendon jerks exaggerated, legs > arms: all abdominals absent; both plantar responses extensor.

Progress: Two months later both legs were more spastic and weaker.

Note: Early case; remissions after acute exacerbations.

Case XXIV.—H. W., man, aged 26, soldier. Admitted to St. Thomas's Hospital on February 3, 1915.

Family and past history good.

History of present illness: Ten weeks ago patient had a seizure and was

unconscious for an hour; on regaining consciousness he complained of severe headache and retro-ocular pain. A few days later he noticed that he was nearly blind in his right eye. He remained in bed for a month owing to severe giddiness every time he moved, accompanied by vomiting. He also had difficulty in holding his water and had to answer the call the moment he felt it. Four weeks ago he noticed that his hands were unsteady, and he also had cramp-like pains in his legs. His general condition has recently improved.

On examination, February 3, 1915: Fair type, good physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition: intelligent; unduly sanguine and distinctly emotional. Vision: acuity, $\frac{3}{60}$ right, $\frac{6}{6}$ left; field in right eye is greatly contracted and there is no colour vision; there is a central scotoma involving the fixation point, spreading out into the temporal field more than the nasal; the field in the left eye is normal; the right disc is pale all over, the edges are slightly blurred, but there is no measurable swelling; the left is normal. Hearing normal; no vertigo now. Cranial nerves: except that the right pupil reacts rather sluggishly to direct light the pupillary reactions are normal; weakness of right external rectus; rotatory nystagmus on lateral movements, especially to the right; otherwise negative. Motor.—Head and neck, nodding tremor, when unsupported. Trunk and limbs: no paresis, hands are a little unsteady, but there is no true intention tremor or inco-ordination; writing is good. Sensory: no objective impairment. Sphincters: urgent micturition. Gait natural. Reflexes: all tendon jerks exaggerated; abdominals present and equal; both plantar responses flexor.

Progress: A month later vision was unchanged. He feels depressed, but laughs when spoken to. Urgency of micturition has increased with several "accidents." Numerous fleeting paræsthesiæ, especially in arms. Gait a little uncertain, but not definitely cerebellar.

Note: Early case; acute onset with loss of consciousness; wide dissemination of lesions.

Case XXV.—C. W., woman, aged 37, married. Admitted to National Hospital, Queen Square, on February 15, 1915.

Family history: Mother is "nervous."

Past history: Always of a nervous disposition.

History of present illness: When pregnant fifteen years ago she noticed that her legs frequently twitched: this has persisted, and she has never been able to walk fast since, although not regarding this as a disability. Ten years ago her left eye rapidly became nearly blind; after a few weeks the sight returned, and she believes it is now quite good. Four years ago she had three severe attacks of rotatory vertigo, associated with vomiting and headache. Three weeks ago, after a thorough wetting, she had severe tingling followed by coldness and numbness extending up the legs to the waist; she also felt as if a tight band were round her waist. These sensations have gradually diminished. Recently she has had slight frequency of micturition, but no urgency.

On examination, February 15, 1915: Good type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition, vision, and hearing normal; visual acuity is full in both eyes and there is no pallor of the discs. Cranial nerves negative. Motor.—Head, neck, and upper limbs normal. Trunk: weakness of muscles below umbilicus. Lower limbs: slight extensor spasticity and weakness of flexors. Sensory.—Subjective: numbness from waist downwards. Objective: no definite impairment; vibration not tested. Sphincters: natural. Gait: spastic. Reflexes: arm jerks brisk; leg jerks greatly exaggerated; upper abdominal reflexes present, lower absent; both plantar responses extensor.

Progress: Six weeks later the gait had greatly improved, and the numbness had almost disappeared.

Note: Remittent type extending over a long period; very little actual disability.

Case XXVI.—R. T., man, aged 29, porter. Admitted to St. Thomas's Hospital, April 23, 1915.

Family history negative.

Past history: Typhoid fever, aged 25.

History of present illness: Nine months ago he began to see double; this has persisted. Five months ago, when out walking, he suddenly became unsteady on his legs and nearly fell down; he had considerable difficulty in getting home. The same evening his left hand began to shake so that he could hardly feed himself. Since then his walking and tremulousness have got worse, and his head shakes when he moves about. During the past month he has been subject to sudden attacks of rotatory vertigo without tinnitus or vomiting, in which he tends to fall to the left. Never dysarthria, paræsthesiæ, or sphincter trouble.

On examination, April 23, 1915: Fair type, good physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition: no loss of emotional control, but undue optimism and indifference to his disabilities. Vision: acuity, right uncorrected $\frac{6}{60}$, corrected $\frac{6}{18}$; left $\frac{6}{9}$; fundi normal, except for myopia in right eye. Hearing good. Cranial nerves: pupils normal; slight ptosis on right, and weakness of right external and internal recti with diplopia; coarse slow nystagmus in all directions; otherwise negative; no dysarthria. Motor.—Head and neck: nodding tremor of head, increased by excitement and when he walks. Upper limbs: no paresis; well marked intention tremor, left > right, with dysmetria and dysdiadokokinesis; all movements clumsy and laboriously slow; no sensory ataxia. Trunk: combined flexion of hip and trunk well shown. Lower limbs: a little extensor spasticity; no paresis; intention tremor, left > right. Sensory.—Subjective: normal. Objective: slight astereognosis in right hand, with lowering of compass threshold (2·5 cm.); no definite loss of passive posture and movement; otherwise negative; vibration normal. Sphincters natural. Gait: tremulous, unsteady and reeling; well marked asynergia;

Rombergism negative. Reflexes: all tendon jerks exaggerated, legs > arms; all abdominals absent; both plantar responses extensor.

Progress: Three months later his walking had enormously improved and his hands were much steadier.

Note: Early case; remittent type; cerebellar manifestations prominent.

Case XXVII.—M. F., woman, aged 21, servant. Admitted to St. Thomas's Hospital on May 19, 1915.

Family history: Both parents, two brothers and one sister all died of phthisis.

Past history good.

History of present illness: Eight years ago her right hand suddenly became useless; she could not grasp things properly nor feel what she held in her hand. Ten days later the right leg was affected and she stumbled when walking. This was shortly followed by the appearance of tremors in the arms, especially the right, which rendered dressing and feeding very difficult. Her speech also was said to have altered. In ten months all symptoms had entirely disappeared and she felt perfectly well. Four years ago she had precisely similar symptoms, though not nearly so severe; after four months she again got quite well. One year ago she noticed curious sensations in the right arm and leg, which began to shake when she moved them; she could not identify objects in the right hand, and her speech became slow and stammering. She improved for a time, but six months ago got much worse and has gone downhill ever since. Her gait has become unsteady, she has difficulty in passing her water, and has become very emotional and unable to control her feelings. Recently she has had attacks of vertigo. No ocular symptoms.

On examination, May 19, 1915: Good type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition intelligent; profoundly emotional and laughs inordinately at her own tremor. Vision: acuity normal; no ophthalmoscopic examination. Hearing good. Cranial nerves: fine nystagmus on lateral movements: typical ataxic dysarthria, slurring, staccato, monotonous, breathless. Motor.—Head and neck: well-marked nodding tremor. Upper limbs: no paresis; well-marked intention tremor and asynergia, right > left; tabetic athetosis of right hand. Trunk: combined flexion of hip and trunk well marked. Lower limbs: no paresis or definite hypertonia; intention tremor, right > left. Sensory.—Subjective: numerous paræsthesiæ. Objective: definite impairment of sense of position and movement in right hand with raised threshold to compasses (3·5 cm.) and partial astereognosis; otherwise normal. Sphincters: hesitancy of micturition. Gait: no spasticity; tremulous, reeling, and staggering: profound asynergia; no Rombergism. Reflexes: all tendon jerks exaggerated: all abdominals absent; right plantar response flexor, left extensor.

Progress: No change four weeks later.

Note: Remittent type; cerebellar manifestations prominent.

Case XXVIII.—M. S., woman, aged 30, married. Admitted to St. Thomas's Hospital on June 14, 1915.

Family history good.

Past history: Always of a nervous disposition.

History of present illness: Nine years ago she had an illness diagnosed as chorea, during which "her right arm shook whenever she used it"; in three months' time she was quite well again. Three years ago she noticed that she frequently laughed and cried for quite inadequate reasons. At about the same time her walking became unsteady, as if she were drunk, and she had difficulty in holding her water, with frequent "accidents." After about twelve months these symptoms disappeared, but eight months ago they reappeared in a somewhat exaggerated form, together with a coarse tremor of the right arm, so that she had difficulty in writing and in feeding herself. Recently she has had attacks of rotatory vertigo without tinnitus, and the unsteadiness of gait, emotionalism, and loss of control over the bladder have become more pronounced. Never ocular symptoms, dysarthria, or paræsthesiæ.

On examination, June 14, 1915: Good type, fair physique. Visceral examination: slight visceroptosis; otherwise negative. Wassermann reaction (blood) negative. Mental condition: intelligent; very sanguine and facile; laughs at her own tremor; "my expression does not reflect my feelings." Vision: acuity, right $\frac{6}{12}$, left $\frac{6}{6}$ partly; fields not tested; whole of right disc pale compared with left, especially in temporal half; left disc physiological. Hearing normal. Cranial nerves: no ophthalmoplegia; coarse nystagmus with lateral movements; no dysarthria. Motor.—Head and neck: to-and-fro tremor when unsupported. Upper limbs: slight weakness on right compared with left; no spasticity; typical intention tremor, right much > left: asynergia and dysdiadokokinesis, right > left. Writing shows pronounced dysmetria. Trunk: combined flexion of hip and trunk present. Lower limbs: slight extensor spasticity on right, with slight weakness of flexors; no definite paresis on left; no tremor. Sensory: subjective and objective, normal in every particular. Sphincters: precipitate micturition; frequent "accidents." Gait: can just walk without help; not obviously spastic, but reeling, titubating and very unsteady; no Rombergism. Reflexes: all tendon jerks greatly exaggerated, right > left; all abdominals absent; both plantar responses extensor.

Progress: Two months later she could walk round the ward without assistance, but was otherwise *in statu quo*.

Note: Remittent type tending to chronic progression in later stages; no sensory disturbances in spite of wide dissemination of lesions.

Case XXIX.—F. O'B., woman, aged 48, dressmaker. Admitted to St. Thomas's Hospital on June 16, 1915.

Family history: One of ten children; first three were stillborn, all others alive and well except patient and a younger sister, who is undeveloped both physically and mentally.

Past history: Always "suffering with her nerves"; neurasthenia twelve years ago with numerous phobiæ and apprehensions.

History of present illness: Ten years ago her "nerves" were bad, the sight of the left eye was affected and she had difficulty in holding her water. She improved after a few months, and remained fairly well for nearly three years. Seven years ago her left arm suddenly became useless; it was not paralysed, but she dropped things without knowing it and could not feel properly; her gait became affected shortly after and she walked as if drunk. In six months time she had completely recovered, but a little later her right arm became weak, she developed a squint in the left eye and saw double, she had frequent attacks of vertigo, her right leg dragged, and she lost control over her bladder. The diplopia and vertigo disappeared after a year, and although her walking improved, the other symptoms have more or less persisted, but her condition has varied a great deal from time to time. One year ago she had a "nervous breakdown," became unduly emotional, and lost control over the rectum. Six weeks ago she suddenly lost the use of both legs completely and has been in bed ever since. No dysarthria or intention tremor.

On examination, June 16, 1915: Neurotic type, good physique. Small superficial bed-sore over sacrum. No pyrexia. Visceral examination: slight albuminuria; otherwise negative. Wassermann reaction (blood) negative. Mental condition: introspective; poor emotional tone. Vision: acuity, right $\frac{6}{6}$, left $\frac{6}{9}$ partly; fields full; right disc normal, left shows temporal pallor. Hearing normal. Cranial nerves: fine nystagmus on lateral movement; otherwise negative. Motor.—Head and neck natural. Upper limbs: slight weakness and spasticity, right and left; no intention tremor or ataxia. Trunk: weak with some rigidity of abdominal muscles. Lower limbs: almost complete spastic paralysis in extension with frequent flexor spasms. Sensory.—Subjective: girdle sensation. Objective: cutaneous sensibility perfect; vibration abolished in both lower limbs and pelvis; no loss of deep sensation in arms or hands. Sphincters: incontinence of urine and fæces; feels "call" and passage. Gait: impossible. Reflexes: all tendon jerks greatly exaggerated: all abdominals absent; both plantar responses extensor.

Progress: Two months later *in statu quo*.

Note: Duration ten years; remittent type; recent complete paraplegia.

Case XXX.—A. K., girl, aged 16. Admitted to St. Thomas's Hospital on June 22, 1915.

Family and past history good.

History of present illness: Five weeks ago in the course of three days she developed shaking of the hands, drunken gait, attacks of vertigo and headache. The left thigh felt numb and she had pins and needles in both feet. During the past week the tremor of the arms has nearly disappeared. No ocular or sphincter trouble.

On examination, June 22, 1915: Good type and physique. Visceral exami-

nation negative. Wassermann reaction (blood) negative. Mental condition, vision and hearing: normal. Cranial nerves: fine, rapid nystagmus on lateral movements; otherwise negative. Motor.—Head and neck normal. Upper limbs: slight weakness of left grip; no spasticity or wasting; coarse static tremor of hands; no true intention tremor or ataxia. Trunk good. Lower limbs: good tone; some weakness of dorsiflexion at both ankles; both limbs show tremor on sustained efforts. Sensory.—Subjective: numbness of both legs, right > left. Objective: normal except for diminution of vibration sense below right knee. Sphincters natural. Gait: a little reeling and lurching; steps unequal in length and direction; no spasticity or Rombergism. Reflexes: all tendon jerks exaggerated; upper abdominal reflexes present, lower absent; right plantar response indefinite, left extensor.

Progress: One month later the gait was almost normal, but the reflexes were unchanged. After-history could not be traced.

Note: Early case; sudden onset tending to recovery.

Case XXXI.—A. C., girl, aged 20. Admitted to St. Thomas's Hospital on June 24, 1915.

Family and past history: Nothing to note; patient is an only child.

History of present illness: Two years ago she suddenly developed a squint in the right eye with diplopia, headache, vertigo, and a tight feeling round the waist. A fortnight later she was well again. Six months later she had a precisely similar attack of the same duration. Two weeks later she had severe pain in the back of the neck and vomited; the following day the the right arm and leg were weak, she saw double, and when she closed her eyes felt as if she were moving round and round. She also had difficulty in holding her head straight, and in passing her water.

On examination, June 24, 1915: Poor type and physique; bad teeth, adenoid facies; pulse and temperature normal. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition and vision normal. Hearing: normal on right; watch heard at 6 in. on left; bone conduction > air; perforation of left drum; no mastoid tenderness; no tinnitus. Cranial nerves: weakness right external rectus with diplopia; fine nystagmus on looking to left, slow and coarse on looking to right; slight tenderness right lower face. Motor.—Head and neck: the head is carried down with chin to the left; the head can be lifted but quickly assumes this "forced" attitude; no weakness or rigidity. Upper limbs: slight weakness with some flexor rigidity of right arm; all movements of the right arm are remarkable for the slowness both of their initiation and execution; attempts at rapid movements slow intention tremor and hypermetria; left arm normal. Trunk good. Lower limbs: slight extensor spasticity on right, with weakness of dorsiflexion at ankles; left normal. Sensory.—Subjective and objective, normal. Sphincters; definite hesitancy of micturition. Gait: spastic weakness of right leg with circumduction at hip. Reflexes: all tendon jerks exaggerated,

right > left ; abdominals absent on right, just obtained on left. Right plantar response extensor, left flexor.

Progress : Two weeks later the attitude of the head was normal, and the right hemiparesis was less marked. The diplopia persisted.

Note : Remittent type with acute exacerbations.

Case XXXII.—F. L., man, aged 37, ex-soldier. Admitted to St. Thomas's Hospital on June 27, 1915.

Family history good.

Past history : Always enjoyed good health. Fourteen years ago he had a soft chancre, for which he was treated, but not with anti-syphilitic remedies ; married ; wife has had two healthy children, and no miscarriages.

History of present illness : Eighteen months ago while out walking the left foot became numb and weak, and his left hand useless, so that he could not distinguish articles in his left hand trousers pocket. The next day he had diplopia, which persisted for two weeks. The condition of the left arm improved, but his leg got worse, his walking became unsteady, and he lost control over his bladder, and to a less extent over his rectum, sometimes having incontinence and at others retention ; he was also unable to feel the passage of urine but was conscious of a "call." Three months after the onset he rapidly improved, and could walk two or three miles without feeling anything amiss in the left leg ; beyond that distance his leg would drag. The sphincter trouble almost disappeared. Nine months ago he relapsed again, his left arm and leg became weaker, he got progressively more unsteady on his legs, and again lost control over his sphincters. He also experienced various odd sensations ("tight feelings") all over his body and limbs, fleeting in character, and became subject to attacks of rotatory vertigo without sickness or tinnitus. A month ago his condition underwent temporary improvement, but his walking is now worse than ever, especially in the dark.

On examination, June 27, 1915 : Good type and physique ; no evidence of past syphilis. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition : vision and hearing normal. Cranial nerves : no ophthalmoplegia ; rather coarse nystagmus on lateral movements ; slight but definite dysarthria, slurring, halting, staccato. Motor.—Head and neck normal. Upper limbs : right normal ; slight weakness of extensors on left, but no rigidity ; no tremor or cerebellar ataxy ; slight sensory ataxy on left with tabetic athetosis. Trunk good. Lower limbs : slight extensor spasticity with weakness of flexors on left ; sensory ataxy of both legs ; no loss of power on right. Sensory.—Subjective : girdle sensation and numbness of left leg. Objective : cutaneous sensibility is lost or impaired down back of left lower limb, sacral segments 1 to 5 ; otherwise normal. Deep sensation : slight loss of passive position and movement in left hand and wrist, compass threshold raised on left (1·5 cm. left palm, 0·75 cm. right palm), partial astereognosis left hand. Moderate loss of sense of position and movement, especially peri-

pherally in lower limbs, left > right, but not sufficient to account for the high degree of inco-ordination. Vibration impaired in left pelvis, but within normal limits elsewhere. Sphincters : difficulty in feeling passage of urine and fæces ; hesitancy of micturition alternating with urgency ; anal reflex present ; absolute loss of sexual power. Gait : can just walk a few steps unaided ; Rombergism present ; the gait is ataxic and reeling and combines the features of both tabetic and cerebellar inco-ordination. Reflexes : all tendon jerks increased, legs > arms. All abdominals absent. Both plantar responses extensor.

Progress : Three weeks later *in statu quo.*

Note : Remittent type with little tendency to the formation of lesions in fresh localities ; involvement of cord in sacral region.

Case XXXIII.—F. T., man, aged 30, policeman. Admitted to St. Thomas's Hospital on July 7, 1915.

Family and past history good.

History of present illness : Two years ago he noticed a mist in front of his left eye, which has persisted. Nine months ago his legs quite rapidly became weak and he had difficulty in getting about. His walking has varied considerably since then, but on the whole it has got worse. Eight months ago he developed urgent micturition with occasional "accidents." More recently he has noticed numbness down his legs and his speech has got slow and jerky. Recently the sphincter trouble has improved ; no "accidents" lately. Never diplopia, tremor, or emotionalism.

On examination, July 7, 1915 : Good type and physique. Visceral examination negative. Wassermann reaction (blood) negative. Mental condition and hearing normal. Vision : acuity, $\frac{6}{6}$ right and left : large central scotoma in left eye, involving fixation point, especially to colours ; left disc is pale all over, edges clear ; right disc normal. Cranial nerves : no nystagmus or ophthalmoplegia ; slight ataxic dysarthria, slow, staccato, and explosive. Motor.—Head and neck, trunk, upper limbs, normal ; no tremor. Lower limbs : slight extensor spasticity and weakness of flexors, left > right ; no ataxy. Sensory.—Subjective, numbness in legs. Objective, normal. Sphincters ; urgent micturition. Gait : spastic ; no Rombergism or ataxy. Reflexes : all tendon jerks exaggerated, legs > arms ; all abdominals absent ; both plantar responses extensor.

Progress ; No material change four weeks later.

Note : Remittent type in early stages tending to become slowly progressive.

Case XXXIV.—C. R., woman, aged 25, married. Admitted to St. Thomas's Hospital on November 16, 1920.

Family history unimportant.

Past history : Since the age of 14 has been subject to attacks of headache and giddiness, with vomiting, diarrhœa and faintness.

History of present illness : Four years ago a few hours after having had

some teeth out under gas she "came over queer" and was unable to see or speak for some minutes: the next day her walking was unsteady. This unsteadiness has persisted off and on ever since, but varies a great deal; for weeks together she will walk as if drunk, and then get nearly well again. She is always much worse in the dark. Three years ago the right hand suddenly became useless; she could move it but could not feel things properly with it. After six weeks it improved, but has never got quite well. Twelve days ago she woke up to find that she was cold and numb all down the left side, and her left arm was useless although not paralysed: the left leg seemed weak and her walking more unsteady than ever. She also felt as if she had a tight band round her waist. Four days later she felt as if hot water were being poured down her left side. Recently she has had attacks of rotatory vertigo. Never speech disturbance, diplopia, or sphincter trouble.

On examination, November 16, 1920: Good type. General condition good. Thoracic and abdominal viscera negative. Wassermann reaction (blood) negative. Mental condition: rather sanguine and optimistic, but no loss of emotional tone; condition is not abnormal. Vision normal in every particular. Hearing normal. Vertigo: objects move from left to right. Cranial nerve normal, no nystagmus. Motor.—Head and neck normal. Upper extremities: no loss of power on right or intention tremor; slight tabetic athetosis of right hand and some sensory ataxia; power on left is subnormal, but with eyes shut this defect is greatly accentuated; wild sensory ataxia on left. Trunk: good. Lower extremities: right normal except for slight spasticity; left, considerable spasticity with weakness, especially of flexors. Sensory.—Subjective: paræsthesiæ trunk and left arm and leg. Objective: cutaneous sensibility, everywhere perfect. Profound loss of deep sensation in left upper limb; she has no idea of the position of the limb and loses it in bed; no threshold to compasses, complete astereognosis; localization perfect, vibration impaired below left elbow. Slight astereognosis in right hand and raised threshold to compasses (2·5 cm.). In the left lower limb there is some disturbance of sense of passive posture and movement with loss of vibration reaching up to the left pelvis; vibration much diminished in right pelvis and lower extremity. Sphincters natural. Gait: spastic and ataxic; Rombergism present: no cerebellar inco-ordination. Reflexes: all jerks increased, especially in legs; abdominals all absent: both plantars extensor.

Progress: Two months later (January, 1921) the condition had greatly improved; the loss of deep sensation in the left arm was diminishing, she no longer loses the limb, and could use a fork in the left hand and dress herself. Reflexes unaltered. April, 1921: improvement maintained; no loss of passive position and movement is demonstrable in left arm, there is no sensory ataxy, and the patient says that this limb is now better than its fellow. The gait is much improved, the right plantar response is flexor and the left extensor.

Note: Remittent type, lesions appear almost confined to spinal cord; remarkable recovery from most recent lesions.

Case XXXV.—L. H., woman, aged 25, domestic servant. Admitted to St. Thomas's Hospital on November 10, 1920.

Family history: Mother very "nervous"; one sister has had chorea.

Past history: Nothing of importance.

History of present illness: Ten years ago she woke up one morning to find that her right arm was useless and her right leg was weak. The leg got well in a few days, but the arm remained bad for four months; it was not paralysed, but felt numb and cold, and she could not use it properly. Ever since she has been subject to paræsthesiæ in the right arm and leg. Six years ago she suddenly developed an internal squint in the left eye and saw double: this condition persisted for three weeks and then passed off. Five years ago after an attack of influenza her legs became weak and her walking unsteady; complete recovery in six weeks. Three years ago her legs began to get weak again, and she had pain in her legs and pins and needles round her ankles. Her walking gradually became worse, so that her feet dragged and she had to go about with the aid of a stick. The weakness of the legs has varied considerably in the past three years, but has never entirely disappeared. Latterly the legs have felt cold and numb. Two months ago she again saw double, and a little later experienced pain behind the eyes, especially the left, which rapidly became blind; the vision is now returning but things "look black out of the left eye." During the past three weeks she has been subject to attacks of rotatory vertigo. No affection of speech or sphincters.

On examination, November 10, 1920: Thoracic and abdominal viscera negative. Wassermann reaction (blood) negative. Mental condition normal, perhaps a trifle sanguine. Vision: right, acuity $\frac{6}{9}$, fields normal, temporal pallor of disc; left, acuity $\frac{6}{36}$, large scotoma, disc edges all blurred, swelling 1·5 D.; no hæmorrhages (retro-bulbar neuritis). Hearing normal. Cranial nerves: pupils normal; weakness left external rectus; well-sustained horizontal nystagmus; otherwise negative. Motor.—Head, neck, trunk, and upper extremities, nothing to note. Lower extremities: slight spasticity, weakness of all flexors; loss of power is greatly accentuated if legs are screened from view during testing; considerable ataxy of tabetic type. Sensory.—Subjective: numerous paræsthesiæ, especially in lower limbs. Objective: cutaneous sensibility everywhere normal; no loss of deep sensation in upper limbs; vibration —complete loss below knees, profound loss in femora and pelvis; sense of passive posture and movement gravely disturbed in lower limbs, especially peripherally. Gait ataxic and spastic; Rombergism present. Sphincters natural. Reflexes: arm-jerks brisk; knee-jerks greatly exaggerated; ankle-jerks diminished; all abdominals absent; both plantars extensor.

Progress: Two months later the vision in the left eye was $\frac{6}{12}$, the scotoma had contracted, and there was no œdema of the disc, which was pale all over. The diplopia had disappeared, the legs were stronger, and the walking had improved. The right plantar response was less definitely extensor than on admission.

Note: Markedly remittent type with exacerbations and recoveries.

REFERENCES.

[1] Adams. *Lancet*, 1921, **1**, 420.
[2] Bullock. *Ibid.*, 1913, **2**, 1185.
[3] Büscher. *Arch. f. Psychiat.*, 1920, **62**, 426.
[4] Charcot. "Diseases of the Nervous System," New Sydenham Society, 1877.
[5] Dawson. *Trans. Roy. Soc. (Edin.)*, 1916, **50**, 517 (complete bibliography).
[6] Dudgeon. *Journ. of Hygiene*, 1920, **19**, II, 208.
[7] Fiessinger. *Compt. rend. Soc. de Biol.*, 1918, **81**, 38.
[8] Holmes. *Brain*, 1904, **27**, 327.
[9] Kuhn und Steiner. *Med. Klinik.*, 1917, **13**, 1007.
[10] Marinesco. *Rev. Neurol.*, 1919, **26**, 481.
[11] Müller. "Die multiple Sklerose des Gehirns und Rückenmarks," Jena, 1904
[12] Oppenheim. "Text-book of Nervous Diseases" (Bruce), 1911, **1**, 332.
[13] Rothfeld, Freund und Hornowski. *Deutsch. Zeitschr. f. Nervenheilk.*, 1921, **67**, 257.
[14] Russell. Allbutt's "System of Medicine," 1911, **7**, 809.
[15] Schuster. *Zeitschr. f. d. ges. Neur. u. Psych.* (Original), 1921, **65**, 1.
[16] Siemerling und Raecke. *Arch. f. Psychiat.*, 1911, **48**, 824; 1914, **53**, 385 (complete bibliography).
[17] Siemerling. *Berl. klin. Wochenschr.*, 1918, **51**, 273.
[18] Simons. *Neurol. Centralbl.*, 1918, **37**, 129.
[19] Steiner. *Zeitschr. f. d. ges. Neur. u. Psych.* (Referat), 1919, **17**, 491.
[20] Strümpell. *Neurol. Centralbl.*, 1896, **15**, 961; 1918, **17**, 401.

THE EXPERIMENTAL STUDY OF DISSEMINATED SCLEROSIS.

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THE history of the experimental study of disseminated sclerosis begins with the publication of a paper by myself [1] in 1913. The paper was a report of experiments in which rabbits and one cat were injected with cerebrospinal fluid, filtered and unfiltered, taken from a rapidly progressing case of the disease. Several of the rabbits became paralysed and the conclusion was drawn that disseminated sclerosis is caused by a filter-passing organism, or by a water-soluble poison present in the cerebrospinal fluid.

The results of the experiments have been confirmed by Kuhn and Steiner [2], Simons [9], Marinesco [5] and by Rothfeld, Freund and Hornowski [7].

Kuhn and Steiner were able to transmit the disease to guinea-pigs as well as to rabbits; in one set of experiments they achieved four successful passages through guinea-pigs and, in another set, two through rabbits. This constituted a notable advance and provided solid grounds for the belief that disseminated sclerosis is an infectious disease. More important still, they observed a spirochæte in the blood of the paralysed rabbits and guinea-pigs. The organism was described as resembling closely the leptospira of Weil's disease; it could be stained with Giemsa's stain and with Loeffler's flagella stain, and was blackened in tissues by Levaditi's silver method. Kuhn and Steiner have put forward the claim that the spirochæte they describe is the essential ætiological agent in multiple sclerosis.

Siemerling [8] observed a similar organism post mortem in the cerebral lesions of a patient who suffered from disseminated sclerosis, but who died of an intercurrent facial erysipelas.

Rothfeld, Freund and Hornowski, in a critical paper, give an account of the transmission in series of nervous disorders in rabbits and guinea-pigs. These authors made an exhaustive bacteriological and histological study of the animals which were killed or which died in the

course of their work. The bacteriological results were uniformly negative; they were unable to find Kuhn and Steiner's spirochæte, though all the usual methods of search were employed. The chief pathological features of their animals were the frequent occurrence of coccidiosis, pulmonary inflammatory lesions, cellular infiltration of the kidneys and of tuberculosis. The failure to find a spirochæte and the frequent occurrence of coccidiosis and of inflammatory lesions led the authors to reject the obvious conclusion that the transmission of paralysis in series—beginning with cerebrospinal fluid as the primary inoculum, and continuing with organ emulsions from affected animals—was evidence of the existence of a living virus in the cerebrospinal fluid; they consider that the close analysis of the anatomical pathological findings—the non-specific character of the changes found, the constantly negative bacteriological results, the fact that the intensity of action of the inoculum gets weaker in successive passages and that in some of their animals disease and death were caused by (or associated with) coccidiosis or tuberculosis—points definitely against the simpler interpretation. The difficulties met by these authors are inevitable in such experiments, and, of course, compel caution in drawing conclusions. But there are other and more likely fallacies in the work, as will be shown later. The substance of the published work may thus be divided into two parts: first, the transmission in series to animals; and secondly, the discovery of a spirochæte in affected animals and the observation of a similar organism in the cerebral lesions of man. The first part only will be discussed in this paper. The second part, together with the histological changes in the affected animals, will be dealt with later.

Experimental.

During the past twelve months I have injected rabbits and in a few instances guinea-pigs with cerebrospinal fluid obtained from twenty-one cases of disseminated sclerosis. All the cases were carefully selected from a large number of patients and each exhibited some or all of the classical signs of the disease, namely, partial or complete paralysis, with Babinski's sign, nystagmus, intention tremors and staccato speech. The cerebrospinal fluid in every case gave a negative Wassermann reaction: for carrying out the test in a number of cases I am indebted to Dr. Paul Fildes.

The total number of rabbits injected was 129, and of guinea-pigs 15. The guinea-pigs remained in good health and none ever showed

signs of paralysis; 17 of the rabbits became ill and paralysed and 112 remained in good health, or if they became ill or died the cause was not clearly connected with the experimental injection.

Of the twenty-one sets of experiments in which rabbits were injected with cerebrospinal fluid—the numbers varied from two to six in the experiments—fourteen were completely negative or, when illness occurred, there were such complications as diarrhœa or extensive coccidiosis. Under such circumstances the experiment was written off as negative.

The seven sets of positive experiments may be divided into groups of four and three respectively. In the first group attempts to transmit the disease in series failed; in the second group transmission for two or more passages was successful. Two of these experiments will be given at length.

Case No. 4.—The patient, K. B., a girl of 18, was lumbar-punctured on August 3, 1920. The cerebrospinal fluid issued under pressure and was clear and colourless; the cell count was not increased.

On the same day four young rabbits and two guinea-pigs were injected with the fluid. Rabbit 1 received $\frac{1}{4}$ c.c. subdurally; Nos. 2 and 3, $\frac{1}{2}$ c.c. into the thigh muscles; No. 4, 1 c.c. subcutaneously. The guinea-pigs were injected intramuscularly and subcutaneously, each with 1 c.c. Rabbits 2, 3 and 4 and both guinea-pigs remained in good health and were killed three months later. On the morning of August 6, Rabbit No. 1 was noticed to be ill and weak in the fore-limbs, the animal was unable to hold up its head, which was wet and soiled with the morning's feed. The animal got rapidly worse and was killed in the afternoon of the same day. Post mortem nothing abnormal was noticed in the thorax and abdomen; the surface of the brain was redder than usual. Cultures were made in broth and serum broth from the heart blood and the brain: they remained sterile for seven days. Cerebrospinal fluid was taken aseptically from the fourth ventricle diluted, and injected subdurally into two normal rabbits. Pieces of brain were excised, minced with sharp scissors and injected intraperitoneally in doses of $\frac{1}{2}$ c.c. into four rabbits.

Of the six rabbits inoculated four remained in good health; they were the two which had been injected subdurally with cerebrospinal fluid and two of the four which had received brain emulsion intraperitoneally. Of the two remaining animals one was found dead on the morning of August 8; extensive coccidiosis was found post mortem and the animal was discarded.

The remaining rabbit remained in good health until August 12, when it was noticed to be quiet. On the following day it was found dead. The course of the illness was thus not observed.

Post mortem.—Heart and lungs were normal. The liver and spleen were small. The inoculum of brain emulsion was found and films made from it were stained for bacteria, and none were found. The surface of the brain was deep pink in colour; on section no hæmorrhages were found. Heart blood culture was negative.

Two rabbits were injected intraperitoneally with an emulsion of brain; one of the animals remained in good health, the other died on September 23.

Post mortem.—Nothing abnormal was found in the thorax: in the abdomen there was a large excess of clear colourless fluid, which clotted after incubation for one hour at 37° C. The surface of the brain was congested.

Four rabbits were inoculated intraperitoneally with the brain; they remained in good health and were killed three months later.

The course of the experiment is summarized in the following chart, where O indicates that the animal remained in good health, + signifies died, and Z means killed.

CHART I.—*K. B.*, AUGUST 3, 1920.

K. B. 1A	Rabbit No.:	1	2	3	4
		S.D.	I.M.	L.M.	Sub.cut.
3.8.20		Z 6.8.20	0	0	0

↓

K. B. 2A	Rabbit No.:	1	2	3	4	5	6
		S.D.	S.D.	I.P.	I.P.	I.P.	I.P.
6.8.20		0	0	0	+	0	+
					8.8.20 (discarded)		13.8.20

↓

K. B. 3A	Rabbit No.:	1	2
		I.P.	I.P.
13.8.20		0	+ 23.9.20

↓

K. B. 4A	Rabbit No.:	1	2	3	4
23.9.20		I.P.	I.P.	I.P.	I.P.
		0	0	0	0

Case No. 6.—The patient (M. K.) was a woman aged 36, who had suffered from disseminated sclerosis for four years. She was unable to walk; the legs were contracted. A lumbar puncture was made on September 3, 1920. The fluid poured out copiously and was clear and colourless. There was no excess of cells; the Wassermann reaction was negative.

On the same day six rabbits and four guinea-pigs were injected with the cerebrospinal fluid. The guinea-pigs remained in good health and were killed after three months. Of the rabbits three were injected into the muscles of the thigh, one was injected intravenously, one intraperitoneally and one intra-ocularly. Only the last rabbit eventually showed any symptoms, the other five remaining in good health.

The animal which had been inoculated intra-ocularly was found ill and paralysed on the morning of September 27; it got worse during the day and was killed in the afternoon. Post mortem nothing abnormal was found in the thorax; in the abdomen, a moderate degree of coccidiosis. Four normal rabbits were injected with brain emulsion.

Of these four rabbits one died during the week-end, October 20, and was thrown away.

On October 27 one was found paralysed and dying; this animal had been distinctly ill from October 20. Post mortem the heart and lungs were normal; the liver was small, the stomach empty, the bladder was enormously distended with urine and the large intestine full of fæces. The brain surface was pink. Heart blood cultures remained sterile for seven days.

Five normal rabbits were injected intraperitoneally with an emulsion of the brain. The course of events in these rabbits may be seen in Chart II, from which it will be noted that all the animals became affected and were killed or died. One, however, No. 5, suffered from diarrhœa and was found post mortem to be extensively affected with coccidiosis. This animal must therefore be disregarded.

The rabbit from which the next series of inoculations was made (No. 1) was ill and tremulous on November 1; the following day it was much worse, unable to raise itself or to move its limbs; the head was retracted. Post mortem nothing abnormal was found in the thorax; in the peritoneal cavity there was a large excess—amounting to 30 c.c. roughly—of clear colourless sterile fluid which clotted on standing at 37° C.

Four normal rabbits were injected intraperitoneally with an emulsion of brain; two of these survived indefinitely, and two died.

The two latter animals were examined post mortem and, as in the cases above, no obvious cause of death could be found. Cultures were made of heart blood, peritoneal exudate and cerebrospinal fluid; all were negative. Further passages were not attempted.

Another passage, 4B, was made from an animal which was killed on November 3, after an illness lasting twenty-four hours Three rabbits were inoculated: one died on November 8, and one on the 12th; post mortem examinations failed to reveal the cause of death; there was an excess of sterile fluid in the peritoneal cavity; the heart blood and cerebrospinal fluid were sterile. The third rabbit was killed on November 25 for another purpose; it had shown no signs of disease.

The experiment is summarized in Chart II.

CHART II.—*M. K.*, SEPTEMBER 3, 1920.

```
M. K.
1A       Rabbit No:   1      2      3      4      5      6
                      I.M.   I.M.   I.M.   I.P.   I.O.   I.V.
 3.9.20               0      0      0      0      ↓      0
                                                Ill 27.9.20 Z
                                                  ↓
                                                M. K.  27.9.20
                                                  2A
                      Rabbit No.:   1      2      3      4
                                    I.P.   I.P.   I.P.   I.P.
                                    +      ↓      0      0
                                    Week-  Ill
                                    end    Z
                                    Oct. 20 Oct. 27
                            M. K., Oct. 27.
                                 3A
Rabbit No.:  1          2          3          4          5
             I.P.       I.P.       I.P.       I.P.       I.P.
             |          +          +          |          |
             Nov. 2     Nov. 3     Nov. 3     Nov. 3     Nov. 8
             very ill                         very ill   (diarrhœa)
             Z                                Z
                                              ↓
              M. K., Nov. 2                  M. K., Nov. 3
                  4A                              4B
Rabbit No.:  1        2        3     4       1        2        3
             I.P.     I.P.     I.P.  I.P.    I.P.     I.P.     I.P.
             +        +        0     0       +        +        0
             Nov. 11  Nov. 12                Nov. 8   Nov. 12  Z Nov. 25
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DISCUSSION.

It has been found that rabbits injected with cerebrospinal fluid taken from cases of disseminated sclerosis have in some instances

developed an illness accompanied by paralysis; in the two sets of experiments reported, one rabbit out of four in one experiment and one out of six in the other became affected. The injection into normal rabbits of minced brain from the paralysed animal has led to the development of a similar kind of illness, and this has been carried through four passages.

As in the experiments of Rothfeld, Freund and Hornowski already referred to, the simplest interpretation of these results is to assume the existence of a living virus, which is pathogenic to rabbits, in the cerebrospinal fluid; the virus becomes located in the brain, whence it may be carried to other rabbits in series.

It would be rash, however, to draw this conclusion from such a small number of experiments without proper controls—which have to be discussed—unless definite ocular or cultural evidence of the existence of an organism in the cerebrospinal fluid could be provided. At best, such experiments on rabbits can lead only to conclusions of probability, though where a *virus fixe* has been obtained, as in the recent work on encephalitis lethargica, the probability becomes a practical certainty. But up to the present a *virus fixe* has not been obtained in work on disseminated sclerosis.

The alternative explanation which Rothfeld, Freund and Hornowski have offered for such results is not more than a remote possibility. The naked-eye post mortem findings in rabbits which have died in the course of the experiments recorded above differ only in the following particulars from what has been found in post mortems made during the same period on normal animals which had been killed for various purposes; namely, the constant slight congestion of the surface of the brain, clear peritoneal exudate, and the occurrence of small hæmorrhages in the spinal cord and brain. That is to say, it has not been possible to explain the illness and paralysis by means of gross anatomical changes or of infections. Where extensive coccidiosis has been found in an experimental common animal, the animal has been disregarded. The existence of diarrhœa, often a sign of coccidiosis, has throughout been regarded as of sufficient importance to warrant rejection of a rabbit. The microscopic evidence of inflammatory lesions in the lungs and kidneys of rabbits is stressed excessively by Rothfeld and his co-workers; such changes can be found in almost any rabbit and in animals killed roughly may amount to naked-eye hæmorrhages [7].

A much more important criticism of all this experimental work can

be founded on the well-established fact that rabbits especially and guinea-pigs to a less extent are prone to spontaneous paralysis. The frequency with which this occurs has, in my experience, varied considerably in different laboratories. But there is no doubt that wherever large numbers of rabbits and guinea-pigs are kept cases of paralysis are observed from time to time. As an illustration of the commonest form the following case will be given in some detail.

A healthy young rabbit (1 kilogram), one of a series of six which formed part of a feeding experiment just begun, was recorded in the laboratory book as in good health at 10 o'clock in the morning; at noon it was found completely paralysed in the hind limbs. The rabbit was not ill, it was anxious for food and responded when touched. At 2 p.m. it was killed with coal gas. Post mortem nothing abnormal was found in the thorax ; in the abdomen a moderate degree of coccidiosis of the liver was noted and the bladder was enormously distended with clear urine. Examination of the spinal column revealed a fracture at the level of the 11th dorsal vertebra: the spinal cord at this level was contused and infiltrated with blood. It was impossible to discover how the rabbit came by the accident.

I have studied the clinical conditions and the post mortem findings of most of the cases of spontaneous paralysis—between twenty and thirty—in normal rabbits occurring in the laboratories of the National Institute during the last twelve months. The principal clinical features of these cases have been, first, the suddenness in onset ; secondly, the paralysis is the principal condition, the rabbits often being otherwise perfectly healthy ; thirdly, the paralysis is always of the hind limbs only ; fourthly, the animals sometimes recover, and when left they may live for weeks or even months until ascending infection of the urinary tract or persistent incontinence of fæces compels one to kill them. Post mortem examination generally reveals some gross morbid condition to account for the paralysis. The commonest pathological conditions which I have found in such animals is fracture of the spine. In two cases an abscess involving the spinal column was found; one was in a guinea-pig and one in a rabbit. Films of the pus were stained for the tubercle bacillus and were negative. A tape-worm cyst of the spine muscles was found in one animal; in another no gross lesions were discovered, but section of the hardened spinal cord showed scattered small hæmorrhages. The clinical features of this last animal were such as have already been described.

In rabbits which become paralysed after the injection of cerebro-

spinal fluid the paralysis is not the dominant feature; it is only part of a severe and general illness, which, as a rule, progresses very rapidly, but may sometimes be detected for several days before the animal is seriously ill. The clinical differences between these cases of experimental paralysis and the spontaneous cases are so great and constant as to lead me to conclude that they are essentially different in origin. The post mortem findings support this conclusion.

Another fact which must be borne in mind in drawing conclusions from the experimental work on disseminated sclerosis is the infrequency of successful experiments. In the series reported above one rabbit only in four in the first experiment and one in six in the second showed symptoms. This result corresponds with what other observers have published. It is, however, not difficult to understand. The organism, if there be one, is not constantly present in cerebrospinal fluid and is never present in large numbers. In tubercular meningitis it is the exception rather than the rule to find tubercle bacilli in the cerebrospinal fluid and in general paralysis of the insane the spirochæte of syphilis is not often found. Moreover, differences in susceptibility are easily brought out when the inoculum contains a small number of organisms.

The necessary control experiments for this work have not yet been carried out, namely, the inoculation of a large number—say 100—of rabbits, with the cerebrospinal fluids of persons certainly not suffering from multiple sclerosis. Simons controlled his work in this way, but the number of his experiments was small.

The effects of the injection of normal rabbit brain into a normal rabbit have been studied by Remlinger [6], who found that in 100 rabbits thus treated—sometimes with "enormous quantities"—only one animal exhibited symptoms of paralysis. Remlinger gives a description of the clinical condition of this animal which corresponds very closely with what has been stated above. His observations serve to exclude the possibility that the second and later passages of the experiments given in this paper are fallacious.

The conclusion drawn from the work is, therefore, that disseminated sclerosis is probably an infectious disease and that the virus may sometimes be found in cerebrospinal fluid.

I am indebted to Drs. A. G. L. Reade, Wilfred Harris and George Riddoch for permission to take cerebrospinal fluid from the patients under their care.

BIBLIOGRAPHY.

[1] BULLOCK, W. E. (now GYE). "The Experimental Transmission of Disseminated Sclerosis to Rabbits," *Lancet*, 1913, **2**, 1185.

[2] KUHN, PH. und STEINER, G. "Ueber die Ursache der multiplen Sklerose," *Med. Klin.*, 1917, **12**, 1007.

[3] "Ueber die Ursache der multiplen Sklerose," *Zeitschr. für Hygiene u. Infectionskrankheit*, 1920, **90**, 417.

[4] MAITLAND, H. B., COWAN, M. L., and DETWEILER, H. K. "Spontaneous and Artificial Pulmonary Lesions in Guinea-pigs, Rabbits, and Mice," *Brit. Journ. Exp. Path.*, 1921, **2**, 8.

[5] MARINESCO, G. "Étude sur l'origine et la nature de la sclérose en plaques," *Rev-Neurol.*, 1919, **35**, 481.

[6] REMLINGER, P. "Accidents paralytiques d'origine médullaire provoqués chez le lapin par des inoculations de substance nerveuse normale homologue," *C.R. Soc. Biol.*, 1920, **83**, 171.

[7] ROTHFELD, J., FREUND, J., und HORNOWSKI, J. "Experimentelle Untersuchungen über die Pathogenese der multiple Sklerose," *Deutsche Zeitschr. für Nervenheilkunde*, 1921, **67**, 257.

[8] SIEMERLING, E. "Spirochäten im Gehirn eines Falles von Multiplen Sklerose," *Berl, klin. Woch.*, 1918, **55**, 273.

[9] SIMONS, A. "Zur Uebertragbarkeit der multiplen Sklerose," *Neurol. Centralbl.*, 1918, **37**, 129.

REPORT ON A CASE OF MYOCLONIC ENCEPHALOMYELITIS OF MALARIAL ORIGIN.

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EPIDEMIC encephalitis, the existence of which was at first denied by certain authors, has ended not only in establishing its existence but also in invading the domain of a large number of organic diseases, as is shown by the report of the Local Government Board and the observations of French authors, notably those of Netter. We admit, and we are right in doing so, that the localization of the virus in different segments of the central nervous system can produce the clinical aspect of Parkinson's disease, hebephrenia, catatonia, chorea, myoclonus, &c. It is evident that even if epidemic encephalitis can simulate in a striking manner Parkinson's disease, as proved by Longues, Sicard, and Pierre Marie, one will always be able to find differences between the signs of the "Parkinsonism" of encephalitis and true Parkinson's disease, as Pierre Marie and Mlle. Levi have shown. As a matter of fact, in spite of the apparent resemblance between "Parkinsonism" and Parkinson's disease, the mode of onset of the former, the cause of the affliction, and the character of the symptoms permit us easily to distinguish the one from the other.

Furthermore, as our knowledge of the disease and the conditions which produce it advances, we shall find with certainty that it is confused with other pathological conditions which do not belong to it, as for example hysteria (Georges Guillain, personal observation), tubercular meningitis; even the myoclonic form of encephalitis, to which von Economo and Sicard have attracted attention, can be simulated by hysteria and different infections, amongst which should be included malaria.

I propose in this paper to bring anatomo-clinical proof in favour of this opinion. I will give first of all a detailed account of my observations of the disease and of lesions which we have found in the nervous system, and will then state the lessons which can be learnt from our anatomical and clinical observations.

The case was that of a woman, aged 26 years, who was taken ill on September 3, 1920, suffering from fatigue, anorexia and fever of an indefinite type. She remained in bed for two weeks, and as her condition grew worse she entered a hospital, where she remained for only a few days. At this time she was feverish and unable to walk, and one noticed that she had slight convulsive movements of the limbs and of the face. On September 27 she was transferred to our wards, and we noticed that the skin was pale, and that she could neither walk nor stand up. Speech was difficult and dysarthric. Lying on her back one noticed in the upper limbs almost continuous involuntary movements, accentuated at the extremities. These movements showed themselves in the form of rapid oscillations, which consisted of flexion and extension of the fingers or abduction and adduction, whilst in the arm and forearm the movements were of a more myoclonic type without displacement of the limbs. From time to time the movements of the upper limbs were quickened, and were transmitted to the trunk, which was agitated by contractions rhythmical in character. On the side of the abdomen there were sharp contractions having the same rhythm as the respirations (20 per minute). Respiration was regular but superficial, and at times the patient took deep inspirations. There were rapid lateral movements of the head, and at the same time rhythmic movements of the jaws, the lower jaw coming together in such a manner that there was a gnashing of teeth. The eyes were moved laterally and vertically so that they appeared as if they rolled in the orbits. The eyelids fluttered rapidly, the facial muscles twitched, and the nostrils dilated and contracted.

In the lower limbs the involuntary movements consisted of rapid sharp contractions localized to the thigh muscles. Movements were less frequent in the muscles of the leg. The big toes were in a state of continuous extension, though from time to time they also showed rhythmic movements.

To sum up, the majority of the voluntary muscles were subject to involuntary movements so rapid that one could not count them ; they were myoclonic in appearance, more accentuated in the extremities of the upper limbs, in the jaws, and in the orbit. Further, the muscles of the trunk showed movements, larger in size and less frequent, which were from time to time transmitted to the arms. Beside these movements, which were accompanied by slight displacement of the segments in which they took place, one noticed very rapid muscular contractions.

The tendon reflexes of the limbs were abolished; the pupils were equal and reacted to light and accommodation; the cutaneous reflex of the abdomen and even the plantar reflex were absent.

At the level of the right parotid gland was a swelling of considerable size which was painful.

The pulse was so rapid that it could not be counted at the wrist; the femoral pulse beat at 150 to the minute.

The patient had a slight rigidity of the neck and a suspicion of Kernig's sign. The limbs showed no contractures. The temperature was higher in the morning (38·6° C.) than in the evening (37·4° C.).

On the day of the entry into hospital (September 27, 1920) we wished the patient to get up for the operation of lumbar puncture and to seat herself in a chair; but during this manœuvre she fell into a kind of syncope, the tremblings ceased and her arms fell inert beside her body.

When she had been placed upon the bed it was noticed that her pulse was imperceptible and that respiration had apparently ceased.

Artificial respiration was performed and her respiration was restored, although the pulse remained very feeble. Presently the tremor reappeared little by little and in a few minutes regained its previous intensity.

During the day and night the patient was in a state of delirium.

Lumbar puncture, carried out in the lateral position, showed ten lymphocytes by division (Nageotte) and the blood an intense mononuclear leucocytosis.

The urine was brown and on boiling a thick cloud of albumin appeared. Vidal's reaction and the Weil-Felix test were negative.

On September 28 the temperature rose to 39° C., and the state of the patient became much worse. Respiration became noisy, and she died suddenly at 2 p.m. Just before her death the involuntary movements described above disappeared.

As the patient died almost suddenly we were not able to pursue all our investigations for the purpose of arriving at a correct diagnosis, but from the clinical aspect, the slight fever, and the indefinite lymphocytosis, we thought that we had to deal with a case of epidemic encephalitis of myoclonic form; but examination of the blood, followed by the study later of the alterations in the central nervous system, revealed our mistake. In fact there was revealed in the blood, in addition to the lymphocytosis, the presence of hæmatozoa and even of crescents (fig. 1).

On macroscopic examination the brain had already attracted our attention by its greyish coloration, and besides one saw here and there, in addition to the hyperæmia, punctate hæmorrhages in the cortex, the corpus striatum, and especially in the grey matter of the spinal cord. The meninges were unaffected, the cerebellum was hyperæmic, and the

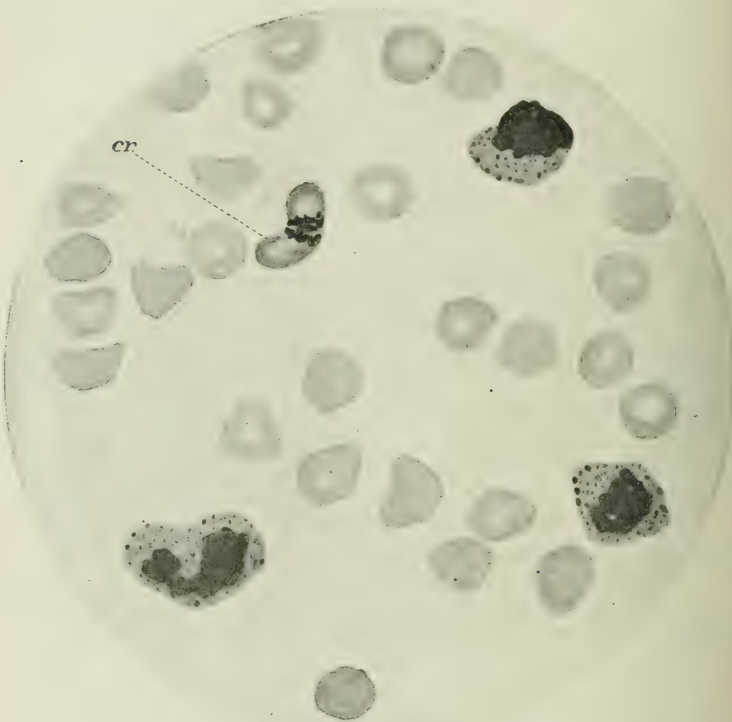

FIG. 1.—Blood-film leucocytes and crescent.

lateral ventricles were slightly dilated. The parotid gland on the right side was congested and showed signs of parenchymatous infiltration. The long narrow vessels which cross the cortex to enter the white matter were dilated and showed, for the most part in their adventitial sheath, a considerable number of lymphocytes, plasma cells, and some

mononuclear leucocytes. Here and there, in the track of precapillary vessels and venules, colonies of plasma cells and fibroblasts were seen, and a similar infiltration of the adventitial sheaths of the vessels was visible in the white matter. Small vessels cut across contained in places a large number of lymphocytes and occasionally also some pigmented mononuclear cells.

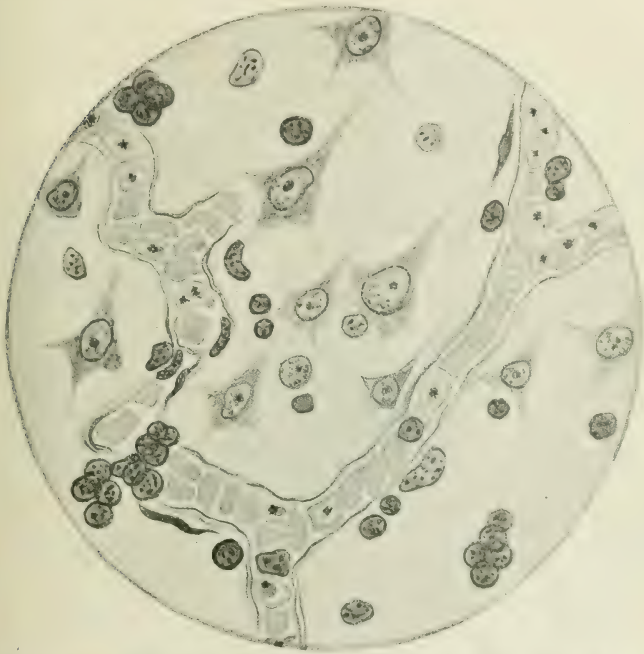

FIG. 2.—Network of capillaries of ascending frontal dilated and, containing in their interior, red corpuscles infected by the parasites and showing pigment.

In general the endothelial cells of the veins, as those of the capillaries, were swollen and sometimes even detached.

The small meningeal vessels were congested and in their sheaths a certain number of lymphocytes and mononuclear cells were recognized.

Hæmorrhages were not the rule. It was quite exceptional to see extravasations in the parenchyma, but hæmorrhagic streaks, which we saw in the spinal cord and of which I will speak later, affected also the

cerebral cortex. Nevertheless I encountered some small hæmorrhages in the cornu ammonis, where we found a thrombus of leucocytes in a meningeal vein, forming a nodule which completely filled the lumen of the vessel.

In all regions of the cerebral cortex infiltrated capillaries and veins were found, dilated or filled with lymphocytes with some mononuclear leucocytes and plasma cells in addition. Throughout on the interior of the vessels red globules were seen which had lost their colour in proportion to the development of the parasite (fig. 2) and a capillary plexus was seen to be distended by these red globules which filled the lumen of the vessel.

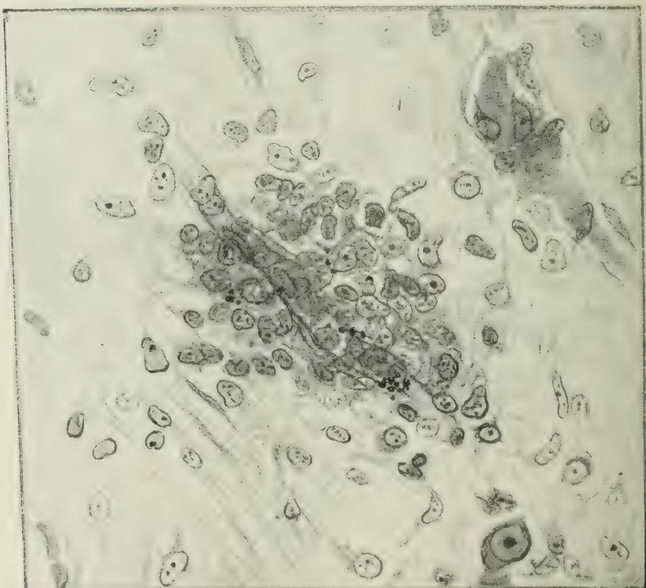

FIG. 3.—Nodule around a capillary in the olivary body; proliferated neuroglia cells.

On the surface of these globules some hæmatozoa, round in shape, were seen occupying them to a greater or less extent.

In the substance of the grey matter, as in the white matter, we remarked an hypertrophy and multiplication of neuroglia cells between

the nerve cells in the neigbourhood of the vessels or near to them. In fact we were able to count up to ten neuroglia cells in the neighbourhood of the base and of the body of the deep pyramidal cells. The same neuroglial reaction existed in the neighbourhood of the small vessels of the white substance, but here we noted a new growth of

FIG. 4.—Small nodule constituted by cells with a clear nucleus and with granular nucleus

neuroglia of peculiar formation caused by the multiplication of these cells in a mass and resulting in a nodule which existed only in the deep white matter in the neighbourhood of the grey matter and which could be found in various sizes. The mean size of these nodules

was 210 μ and 130 μ and they appeared to be formed almost entirely of neuroglial cells.

But there are two reservations which must be made at this point—firstly Nissl's method is not at all satisfactory for the study of the

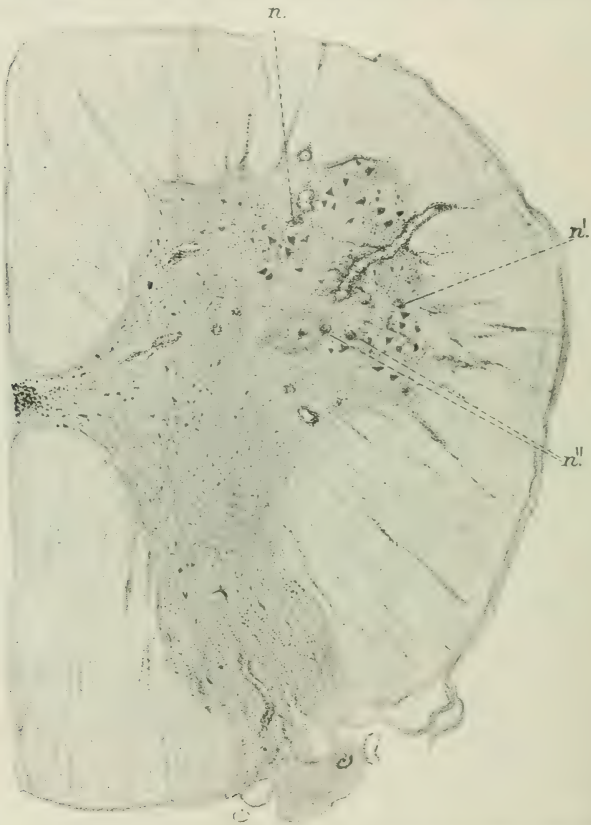

FIG. 5.—Lumbar spinal cord showing infiltration of the vessels in the grey matter and their ramifications and presence of nodules n, n', n''.

histological formation of nodules, and secondly these new growths show a different structure in the brain, peduncle, pons, bulb (fig. 3) and the spinal cord (figs. 4 and 5). One can, nevertheless, distinguish

two species of them in the nervous centres, those of loose structure in which neuroglial cells predominate and others more compact in close contact with the vessels, made up of lymphocytes, mononuclear cells and fibroblasts, to which are occasionally added some plasma cells; at their periphery are a certain number of neuroglial cells, which appear to be increased in size but not multiplied to any great extent.

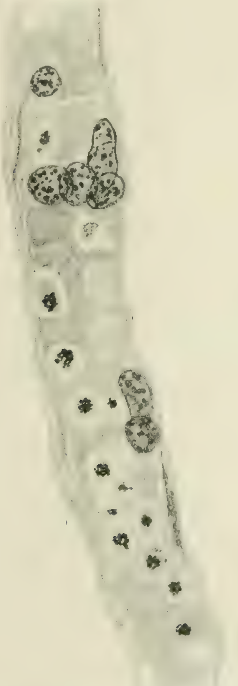

FIG. 6.—Longitudinal section of a small vessel of the grey substance of the bulb, showing in a group of lymphocytes and throughout its extent red corpuscles which have lost their colouring matter owing to the presence of the parasites.

Employing the method of Cajal for neuroglia we have unfortunately not come across (in the sections which we have examined) the nodules we found by Nissl's method; but we stained by this method some sections from a case of exanthemic typhus, and noted that, contrary to

the opinion of some authors, neuroglia played there a rôle which was entirely secondary.

Lesions almost analogous to those in the cortex were found in the optic thalamus, the lenticular nucleus and the caudate nucleus. In the peduncle the infiltration of the vessels and the hyperæmia were more marked in the grey substance than in the white matter and the infiltration of lymphocytes and plasma cells was confined much more to the walls of the veins than to those of the arteries. The cells of the substantia nigra were not particularly affected in spite of the fact that there were more capillaries filled with blood in this region.

On the other hand the same observations are applicable to the red nucleus. The capillary vessels of the corpora quadrigemina and of the grey matter of the peduncles and the promontory (protuberance) showed inflammatory reaction of the small vessels as intense as that in the cerebral cortex.

Very often the capillaries contained hæmatozoa few in number, but there were some of them in which nearly every red globule was parasitic (fig. 6). There were capillaries or precapillaries filled almost exclusively with lymphocytes, small or medium sized, arranged in one or two series. Less frequently these vessels were filled exclusively with polynuclears ending with one or two lymphocytes. In other vessels there was a mixture of lymphocytes and mononuclears and these can predominate.

The nodules which we have described in the white matter of the cerebral cortex are found more rarely in the peduncle. All the lesions which we have just noticed as being in the cerebral cortex, basal ganglia and peduncles are reduced to their simplest expression in the cerebellum when infiltration of the adventitia is exceptional, capillary congestion little marked and the number of hæmatozoa very limited.

We examined for the presence of hæmatozoa different glands such as the spleen, liver, suprarenal capsules, and ovaries, but we have not actually found the parasite of Laveran. There was certainly in the spleen and liver a great quantity of black pigment in the leucocytes and macrophagocytes which filled up the vessels, but I am sure that if there had been hæmatozoa in abundance as we had seen them in the nerve centres it would have been easy to make them evident. So we are obliged to admit that in this case the hæmatozoa localized their action principally to the nerve centres such as the brain, bulb and spinal cord, whilst the cerebellum was invaded to only a slight extent and the spinal ganglia still less.

Other observers before us, Laveran, Councilman, Marchiafava, Monti, Babès, Marinesco, and more recently Lafora. Margulis Cevetti and Babès have described the presence of hæmatozoa in the central nervous system ; but the inflammatory reaction of the vessels and the thrombus of lymphocytes are lesions which have been encountered exceptionally only. As to the inflammatory nodules which we may call by the name "malarial nodules," they are not mentioned in the writings of French, Italian, English or American authors. The essential cause of them seems to be a formative irritation due to the presence of hæmatozoa or their toxins. As to the mechanism of the myoclonic movements it is difficult to venture an exact opinion in the present state of our knowledge.

THE DOUBLE INNERVATION OF STRIATED MUSCLE.

BY PROFESSOR J. T. WILSON.

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THE innervation of the striated musculature may be numbered amongst the many problems which have turned out to be far less simple than they appeared to be to an earlier generation of investigators. This is true whether we have regard to the structural or to the functional side of the problem.

It is the purpose of this brief communication to give an outline of certain contributions, from the morphological side, to our knowledge of the peripheral motor innervation of striped muscle, which are of interest, and possibly of great importance for the physiologist as well as the anatomist. Some of the facts and observations to be detailed have from time to time attracted attention under the heading of a "double innervation of striped muscle." Such a title, however, is ambiguous and unsatisfactory.

In the first place it obviously ignores the distinction between the motor and sensory innervation of the muscle tissue. But even if we substitute the expression "double motor innervation of striped muscle" there remains the objection that two very different conditions as regards the motor nerve supply of muscle have been recognized, either of which may well enough be brought under the category of a "double motor innervation."

(A) PLURISEGMENTAL INNERVATION.

It has long been known that single muscles are commonly supplied by motor nerve fibres from more than one segmental nerve. Indeed the cases of unisegmental nerve supply of a muscle are comparatively few in number. This fact has frequently been interpreted in terms of a corresponding plurisegmental, or polymeric, origin of the muscles so innervated. But the validity of the assumption that all such muscles are necessarily polymeric has not been established beyond doubt. Indeed in the case of the mammalian limb musculature, a derivation of the premuscle blastema from actual embryonic myotomes has not been proved. A definite and obvious origin of the limb muscles from the

myotomes lying opposite the site of origin of the limb buds has indeed been confidently alleged to occur in submammalian forms and it is very likely that the lack of evidence of a similar derivation in mammals is due to cænogenetic blurring of the outlines of the myogenetic process. However that may be, the fact remains that whether they are themselves polymeric or not, a large majority of the muscles of the body are plurisegmental as regards their nerve supply.

The question then arises how the terminals of the various nerve fibre groups supplying a muscle are distributed within the muscle. Are the nerve-fibres from each of the segmental nerves supplying the muscle distributed exclusively to individual muscle-fibres which belong morphologically to the corresponding body segment? Or is there a mutual interpenetration of segmental motor territories on the part of adjacent serial segmental nerves?

In considering these questions it must, however, be borne in mind that the histogenetic study of striped muscle has shown that individual muscle-fibres may be themselves composite segmentally. It has been shown that the myofibrillar differentiation by which the body of the muscle fibre is constituted takes place in a myoblastic syncytium. In the case of adjoining body segments this syncytium forms a continuum which connects contiguous myotomes across the intervening myosepta. Hence a muscle-fibre arising by myofibrillar differentiation in this syncytium may itself be strictly polymeric.

Agduhr (1916-1919) has recently attempted to answer some of the questions regarding the plurisegmental innervation of striped muscle which arise out of the facts outlined above. This author's investigation of the problem arose incidentally from his study of the physiological effects of conjoint and of separate stimulation of two segmental sources of nerve supply of one and the same muscle. Contrary to his expectation he had found that the force of contraction resulting from maximal stimulation of both nerves supplying a given muscle was substantially less than the sum of the contraction force exerted by the muscle when its two sources of nerve supply were separately subjected to maximal stimulation. This phenomenon he was constrained to interpret in terms of an overlapping of the innervation regions of the separate nerves within the muscle. He found also that the region of a muscle thus doubly innervated from two *segmental* nerves was much greater than the region doubly innervated by different *peripheral* nerve paths like the median and ulnar nerves. In other words, the segmental overlap was considerably greater than that of the peripheral nerves, just as

has been found in the case of sensory overlapping. The extensive motor segmental overlap seemed to the author to indicate that a large number of the individual muscle-fibres possess a double, plurisegmental innervation. This question he proceeded to examine by a series of experimental observations.

By the employment of various forms of metallic impregnation (gold chloride as well as his own modification of Bielshowsky's technique) he was enabled to detect and to discriminate between the products of post-operative neuraxon degenerations of varying duration. The following experimental procedure gave instructive results: in a cat the author resected, at different periods, the first thoracic nerve root (ninety hours before the death of the animal) and the seventh cervical nerve root (fifty-eight hours before death). On killing the animal, after the lapse of the period indicated, the M. flexor sublimis digitorum, whose nerve supply is from the seventh and eighth cervical and the first thoracic nerve, was treated by an impregnation method to bring out the motor nerves and their terminations. The three categories of nerve structure corresponding to the three segmental nerves supplying the muscle could be readily distinguished. The endings of the fibres of the eighth cervical nerve were normal. Those of the first thoracic nerve were distinguishable by their more advanced, and those of the seventh cervical nerve by their less advanced degenerative characters. By these criteria it was possible to recognize that many of the individual muscle-fibres were certainly innervated by two successive segmental nerves.

It is well known that one and the same muscle-fibre often possesses two or three motor end-plates. It is therefore interesting to note that this author never came across a muscle-fibre (in the special degeneration preparations) which showed more than one motor end-plate of the same phase as regards degeneration. He will not venture to affirm it as invariable, but suggests that where a striped muscle-fibre has more than one motor end-plate these belong to different segmental nerves.

As against this view, it must be noted that both Floresco (1904) and Cavalié (1901) have definitely described motor nerve-fibres giving off terminal filaments to two or three motor end plaques situated in some cases on one and the same muscle-fibre and in others on neighbouring muscle-fibres or on an adjoining neuro-muscular spindle.

Although these facts would appear to be easily adaptable to the theory of an accurate correspondence between nerve and muscle segment, Agduhr does not favour the view that the plurisegmentally

innervated muscle-fibres he has observed do themselves actually originate from several myotomes. This he regards as unlikely on account of the fact that, as a rule, the end-plates belonging to the different segmental nerves are not placed so far apart on the muscle-fibre as might be expected on such an hypothesis, but, on the contrary, are rather close together. Also he frequently found two closely adjacent end-plates innervated by the seventh cervical and first thoracic to the exclusion of eighth cervical. In his second (1919) paper on the same subject the author has considerably extended his experimental observations confirming his previous conclusions. In order to meet criticism of his earlier work to the effect that the periods he had allowed for degeneration were not such as to exclude mere differences in the degenerative reaction to section of different fibres, Agduhr substantially increased the periods of differential degeneration, and his figures convincingly support the validity of the claim to recognize fibres of different degeneration phase. Figs. 1 and 3 of his paper very clearly illustrate bi- and tri-segmental innervation respectively of individual muscle-fibres.

B.—"Accessory" System of Motor Fibres of Alleged Sympathetic Origin.

An entirely different type of "double-motor innervation of striped muscle" may next be considered. This double innervation is constituted by the presence in the various skeletal muscles so far examined, of non-myelinated motor nerve-fibres which are alleged to be quite independent of the ordinary myelinated motor nerve-fibres not only in structural connexion but in origin and source. These "accessory" motor nerve-fibres sometimes terminate in the same motor end-plates in which the terminals of the ordinary motor nerve-fibres end. But they frequently end in accessory, independent, motor end organs of characteristically smaller size.

Various observers have concluded that these accessory nerve-fibres are of sympathetic origin. This interpretation seemed at first to rest upon slender and unconvincing evidence. But the more recent observations, more especially those based upon the appearances met with as the result of experimental nerve degeneration, seem to establish beyond reasonable doubt at least the independence of the accessory fibres from the system of myelinated fibres which constitutes the hitherto accepted motor supply of the muscle.

The idea of the existence of an accessory, sympathetic source of

motor supply to striped muscle is not novel, although it is only within the last decade that the evidence offered has become strong enough to claim serious consideration.

So far back as 1882 Bremer recorded the discovery, in the frog and the lizard, of fine non-medullated nerve-fibres entering the motor end-plates which form the terminal apparatus of the ordinary medullated motor nerve-fibres of striped muscle. Bremer (1882) distinguished in striated muscle nerve-fibres of three orders: (1) coarse myelinated, (2) fine myelinated, and (3) unmyelinated. He held, however, that the third were connected with the second, though not with the first.

It was not until further advances had been made in neuro-histological technique, as well as in the development and application of the neurone theory, that any significant progress was made in the recognition and discrimination of different categories of nerve-fibres innervating striped muscle.

The introduction of the *intra vitam* methylene blue method of Ehrlich led to many researches on both the sensory and motor innervation of muscle in the late 'eighties and early 'nineties. But neither Gerlach (1889), Dogiel (1891), nor Retzius (1892), all of whom employed this method, has recorded any evidence of specific distinction among the motor innervating nerve-fibres. Nor has Kallius (1896), in his review of this area of investigation, anything further to record in this direction.

Not till we reach the earlier years of the present century do we find further evidence to confirm, and greatly to extend, Bremer's early observations. This newer evidence has been the result of the application of various methods of technique.

These have included modifications of the familiar and well-tried gold chloride method; the newer reduced silver methods of Ramón y Cajal, and, especially, that of Bielschowsky in one or other modification.

Perroncito (1901) described and figured in the lizard unmyelinated nerve-fibres in striated muscle connected with the ordinary motor end-plates but otherwise distinct from the medullated nerve-fibres which end in these plates. Neither the description nor the figures, however, would suffice to differentiate them from the fine, unmyelinated, "ultraterminal" filaments as described and figured by Ruffini (and Apathy) (1900). These ultraterminal fibres are merely unmyelinated filamentous continuations of the neurofibrillar apparatus of an ordinary motor end-plate. Boeke's (1909) more recent investigation of the

development of motor end-plates serves completely to explain in this sense this " ultraterminal " relationship.

In a later paper Perroncito (1902) deals further with the appearance of unmyelinated fibres in muscle. Several instances are there given and illustrated of fine non-medullated fibres accompanying a medullated fibre and ending in the same motor plaques with the branches of the latter. But there is no evidence that the fine fibre shown, e.g., in his fig. 3, Plate I, is not a non-medullated collateral, similar to that actually shown springing from a medullated fibre in his fig. 6, Plate I. Figs. 1, 2, 4 and 5 all show fine non-medullated fibres some of which are obviously collaterals, whilst all may either be collaterals or ultraterminal filaments. Fig. 2 shows a non-myelinated fibre reaching its destination, a motor end-plate, from a different direction to that of the medullated fibre going to the same plate. Probably this is not a collateral from the latter, but there is nothing to show that it may not be an ultraterminal fibre like those illustrated in the text-figures of the author's earlier paper.

Although Perroncito suggests a sympathetic character for the unmyelinated filaments the only evidence adduced is an alleged connection in a few cases with a perivascular nerve plexus. It is clear that neither in the case of the non-medullated collaterals nor in that of the ultraterminal filaments can there be any question of a sympathetic origin or nature of the fibres concerned.

Grabower (1902) contributes the result of his observations on human muscle. He could not recognize any clear-cut distinction of nerve-fibre types, there being all grades from very fine pale fibres to thick medullated ones. He only rarely met with wholly unmyelinated fibres which could be traced as independent fibres for any long distance. Usually they were fibres resulting from the branching of medullated fibres. These often accompanied the latter in a common Henle's sheath, but might end independently.

Gemelli's (1905) observations confirmed Perroncito's, but by showing a definite continuity in the motor end-plates between the arborizing filaments of the unmyelinated fibre and the ramifications of the axon of the medullated fibre, they tended to establish the " ultraterminal " nature of that form of unmyelinated fibre.

Botezat (1906) described a double innervation of striped muscle fibre in birds by both medullated and non-medullated fibres. This author directed attention to the differences between, and the apparent independence, of the two systems. He pointed out that the endings

of the non-medullated fibres were unlike any known sensory ending and resembled the motor, but he did not otherwise determine either their character or their derivation.

Thus far, then, no satisfactory proof of a dual system of innervating motor-fibres had been provided. The appearances which had given rise to the suggestion of such a duality of innervation were probably for the most part to be explained in a quite different and much less remarkable fashion. Nevertheless the suggestion of an anatomical basis for a dual innervation was immediately caught up by some physiologists as providing an hypothesis which some of the physiological phenomena of muscular activity seemed to demand.

Floresco (1904) resected on one side the cervical sympathetic but found no motor atrophy or asymmetry of facial muscles and the motor end-plaques were found to be unaffected in the masseter muscle.

Mosso (1904) quotes Bottazzi's (1897 and 1901) view that the sarcoplasm is the contractile substance which is active in the slow tonic phase of muscular activity, whilst the anisotropic substance is involved in the rapid form of contraction. Mosso, however, denies that the prevalence of tonus in muscle is proportional to its richness in sarcoplasm as Bottazzi was disposed to believe.

It is chiefly the work of Boeke from 1909 onwards which has led to the wide, though by no means universal, acceptance of the evidence for a double motor innervation of striped muscle in the sense now under consideration. In the course of an investigation published in 1909, on the development of the motor end-plates of invertebrates, Boeke was led to recognize the occurrence in muscle of a system of fine non-myelinated fibres which appeared to him to be quite independent of the ordinary myelinated fibres. These non-medullated fibres showed characteristic terminations in the shape of fine end-rings, loops or nets. They were sometimes embedded in the sarcoplasm of the end-plate connected with an ordinary myelinated motor-fibre, but in other cases they ended in small independent end-plates on the muscle-fibres. Boeke laid stress upon their hypolemmal position and their often intimate association with the ordinary motor end-plates as witnessing to their centrifugal and quasi-motor function. He named these non-myelinated fibres "accessory" fibres, and their terminals "accessory" nerve-endings.

In a more extended paper in 1911 and again in 1913, he compared his observations on the "accessory" fibres with the descriptions given by previous observers of non-myelinated filaments noticed by them in

striped muscle. He concludes that for the most part these latter were either simply non-medullated collaterals or similar ultraterminal prolongations of ordinary motor-fibres and were, therefore, not the same as the "accessory" system of fibres discovered by him. He gave definite expression to the opinion (1913) that the "accessory" system of fibres could hardly be other than sympathetic or autonomic in character. Whether they were concerned with the conduction of trophic stimuli or with the tonic innervation of the striated muscle, he left to the physiologists to decide. In this connection accordingly he made reference to the work of Pekelharing and van Hoogenhuyze (1910), who had claimed to establish the occurrence in muscle of two distinct chemical processes, one characteristic of tonic activity and the other of ordinary contraction. He also referred to the support given by Mosso (1904) to the theory of double innervation from the physiological side, and to the more recent work of de Boer (1915) in van Rynberk's Laboratory, where this author claimed to have established experimentally in the case of the frog's muscle that the tonic innervation of muscle is conducted from the spinal cord not along the spinal motor-fibres but along fibres from the sympathetic chain, which reach the mixed nerves through the communicating rami.

In the paper just referred to, de Boer claims to have found that the tonus of most skeletal muscles is carried by the thoracic autonomic system. De Boer also refers to the experiments of Ken Kure, Tohei Hiramatsu and Hachiro Naito which appeared to show that section of the phrenic nerve did not abolish tonus of the diaphragm though it paralysed it; on the other hand, section of the splanchnic nerves produced definite atony of the diaphragm. Following up the physiological side of this question, Dusser de Barenne (1917) was able partially to confirm de Boer's work. He found, however, that the disappearance of tonus of the muscles of the lower limb, after extirpation of the abdominal sympathetic, was not total but partial—hypotony, not atony, was the result—and further, the hypotony gradually disappeared. He concludes, therefore, contrary to de Boer, that the greater part of the muscular tonus is after all conducted by cerebrospinal fibres. He is unable to decide the question whether that part of the tonus which disappears on the production of an acute sympathetic lesion is actually due to the cutting out of autonomic fibres or not. Kuno, working in Starling's Laboratory in University College, London, did not find any diminution of tonus after extirpation of the sympathetic or after section of the rami communicantes.

In a later contribution, Dusser de Barenne (1919) again attacks the physiological problem of the peripheral path for tonic nerve impulses. Here he brings further proof, as against de Boer, that the sympathetic fibres have nothing to do with the transmission of " mechanical muscle tonus." On the other hand, he fully concurs in Boeke's conclusions respecting the sympathetic, centrifugally conducting, character of Boeke's "accessory" fibres, and goes on to suggest that although they can have nothing to do with the propagation of the stimuli of "mechanical" tonus, they may very well be the pathway along which the "chemical" tonus of striped muscle is influenced by the central nervous system.

On the whole it may still be said that the physiological evidence on this question is of a rather equivocal character.

But the morphological evidence not only in favour of the independent existence of Boeke's "accessory" system of fibres, but in favour of its sympathetic derivation, was later supplemented by various observations of the results of experiments in nerve degeneration. Boeke's own experimental results in this direction are embodied in his important "Studien zur Nervenregeneration," I and II, in the *Transactions of the Royal Academy of Sciences of Amsterdam*, in 1916-1917. The nature of the evidence thus obtained may be indicated as follows: In one series of experiments the muscles of the eyeball were investigated. The work of Sherrington, and of Sherrington and Tozer, had shown that the eye muscle nerves in their course from the brain to the muscles form the pathway for all the ordinary muscle-innervating fibres, sensory as well as motor. In a number of experiments Boeke (1917) resected one or other of the oculo-motor nerves close to its origin from the brain. When such an animal was killed a few days (three to five) after the experiment, and a study of the ocular muscles carried out with the aid of the Bielschowsky method, it was found, as was to be expected, that all the medullated nerve-fibres were undergoing degeneration. This was also the case with their associated end-organs. The degenerated fibres and terminals no longer showed the Bielschowsky silver reaction, and this served to bring out with almost diagrammatic clearness the system of non-medullated "accessory" fibres and their hypolemmal endings in the muscle. This system of fine non-medullated nerve-fibres was still so well represented in the muscle, that apparently each muscle-fibre possessed at least one "accessory" platelet. In one of his accounts of these results Boeke suggests that they can only be explained on the hypo-

thesis that these undegenerated, non-medullated fibres are transferred to the eye-muscle nerves by way of the sympathetic branch, which reaches the orbit from the carotid plexus. But his own further experimental observations show that the conditions in this special case of the eye-muscles are not so simple and definite as the account summarized above would suggest. In a series of further experiments Boeke found that when a longer period was allowed to elapse, e.g., three weeks, between the experimental nerve section and the killing of the animal, the greater number—though still not all—of the non-medullated fibres were found to have also undergone degeneration and to have disappeared from the picture. The only explanation offered is that non-medullated fibres resist degeneration after section longer than medullated. On the basis of these facts Boeke suggests that the tardily degenerating non-medullated fibres belong to the cranial autonomic system of Langley included in the eye-muscle nerve-trunks, whilst the other non-medullated fibres—fewer in number—which show no degeneration after a prolonged period following section of the nerve trunk near the brain, are those which have been transferred from the sympathetic to the eye-muscle nerve during its course peripherally.

The attempt to test this result in other cases by extirpation of the superior cervical ganglion showed that this lesion did not lead to disappearance of the system of accessory non-medullated fibres in the eye-muscles, although Boeke records his impression that they were less abundant than in the series with intact superior cervical ganglion. Boeke himself summarizes his conclusion from these facts and observations in a statement which may be translated as follows: "There is present in the eye-muscles, in addition to the motor and sensory nerves, a system of non-medullated fibres independent of these and provided with small hypolemmal end-platelets upon the muscle-fibres. The great majority of these fibres run in the trunks of the motor-nerves of the ocular muscles and belong to Langley's cranial autonomic system; only a small part of the fibres in question belong to the sympathetic in the narrower sense." (1917, p. 18.)

It seems to the writer that Boeke's interpretation is open to grave criticism in more than one direction. Much is made to depend upon the validity of a distinction founded upon mere difference of degeneration time of two categories of fibres. His clearest demonstration of the non-medullated fibres in the ocular muscles is provided by material in which, as it turns out, most of these fibres have already, according to his interpretation, been actually severed from their trophic centres.

They have, therefore, already entered on the career of degeneration. Can one, in these circumstances, be perfectly confident that very thin sheaths of myelin have not already suffered? Again, it is possible that Boeke may be right in identifying these tardily degenerating non-medullated fibres within the muscle with Langley's cranial autonomic fibres, but if they are they ought to be post-ganglionic axones of distribution. Where, then, does the synapse occur in the case, say, of trochlear fibres for the superior oblique muscle? Why, too, it may be asked, should degeneration occur at all if only pre-ganglionic fibres in the nerve-trunk close to the brain have been cut? These latter should of course be fine medullated fibres, if they represent pre-ganglionic elements of Langley's cranial autonomic system.

Further, Boeke does not definitely claim to have shown that the particular non-medullated fibres in the eye-muscles, which he alleges to persist permanently after section of the main nerve-trunk, and regards as sympathetic, have been certainly traced to hypolemmal end-organs. In fact, he admits that he was unable to find degenerated fibres and end-plates in cases where the superior cervical ganglion had been extirpated but the cerebral nerves left intact. There is, therefore, no stringent proof that true sympathetic fibres in the case of the eye-muscles do actually innervate accessory end-platelets in muscle-fibres.

It is strange that in a later paper published by Boeke and Dusser de Barenne in 1919, where the results of the above experiments on the eye-muscle nerves are again referred to, no reference whatever is made to the belated degeneration of the majority of the non-medullated fibres after experimental section. The unqualified statement is made that "the accessory non-medullated fibres, however, and their end-organs on the muscle-fibres remained unaltered, which could only be explained by admitting that they are transferred to the eye-muscle nerves by way of the sympathetic branch," &c. The discrimination introduced two years before between two different groups of non-medullated fibres held to be of diverse origin is now altogether ignored. Fortunately for Boeke's general interpretation the joint paper referred to furnishes other experimental evidence which appears much less open to criticism than that provided by the case of the eye-muscles.

The facts are these: In a cat the anterior and posterior roots of the sixth to ninth thoracic nerves inclusive were resected and the corresponding spinal ganglia were also extirpated. After the lapse of a period of one month the animal was killed and the condition as regards innervation of the muscles of the seventh intercostal space was

then investigated. The result was that while not a single medullated nerve-fibre remained intact and the motor end-plates in connection with them had disappeared there were still present fine bundles of non-medullated fibres connected with the muscle-fibres by means of small and delicate end-organs, end-rings, loops or end-nets. These, as figured in the paper, are precisely like those so abundantly illustrated in previous publications regarding the accessory fibres, and there can be no reasonable doubt of their identity with them.

That the end-organs of such non-medullated nerve-fibres are actually hypolemmal and therefore almost certainly motor in character, is supported by quite a number of Boeke's figures from his various papers, especially those showing profile views of the end-organs in question, e.g., "Studien zur Nervenregeneration," II, 1917, plate 1, fig. 7. The hypolemmal position, to which great importance must be attached, is also convincingly testified to by fig. 2 in the short paper by Boeke (1910), on pp. 481-4 of the *Anatomischer Anzeiger*, Bd. 35. In this figure we have an "accessory" terminal fibre ending in an ordinary motor plate along with the more extensive fibrillar expansion of an ordinary motor-nerve termination. Here, within the end-organ itself, a branch of the "accessory" terminal is seen to pass *under cover of* the fibrillar network of the ordinary motor ending, and therefore could not possibly be other than hypolemmal if honestly and accurately delineated.

In the proceedings of the same meeting of the Royal Academy of Sciences at Amsterdam, at which the paper by Boeke and Dusser de Barenne was presented, there also appeared a paper by E. Agduhr (1919) bearing the title, "Are the Cross-striated Muscle-fibres of the Extremities also innervated sympathetically?" This author investigated muscles of the extremities, especially the interossei muscles in the cat. He performed two different series of experiments. In one series (two cats) he extirpated the ganglion stellatum of one side. On killing the animal after the lapse of a few days he succeeded in finding in the muscle the remains of degenerated non-medullated nerve-fibres. The other series of experiments is more fully reported. They resembled those of Boeke and Dusser de Barenne. He resected the last four cervical and the first two thoracic nerves, in each case at a point between the spinal ganglion and the connection of the nerve trunk with the white ramus communicans. The animals were killed from five to ten days after the operation. On investigating the interossei muscles by the Bielschowsky method it was clear that all the myelinated fibres, both motor and sensory, had undergone degeneration. On the other

hand, the investigator found quite a number of intact non-medullated fibres, and he was able to follow a large number of these to their terminal organs. These were situated partly on ordinary striped muscle-fibres and partly on muscle-fibres in muscle spindles. Fig. 2 of his paper illustrates two ordinary motor end-plates, both degenerated, and situated quite near one another on a muscle-fibre. In one of these degenerated end-plates is seen the undegenerated termination of one of Boeke's "accessory" fibres. Fig. 4, on the other hand, shows in close proximity to one another, on the same nerve-fibre, a degenerated end-plate and an undegenerated one pertaining to the terminal ramifications of a non-medullated, "accessory," "sympathetic" nerve-fibre which are clearly visible in it. Agduhr's results accordingly serve to corroborate in all essentials the observations of Boeke on the independent existence of an accessory system of nerve-fibres. It is therefore difficult, if not impossible, to avoid the conclusion drawn by Boeke and supported by Agduhr and others from the results of what would appear to be crucial experiments, that these accessory fibres are sympathetic in origin and hypolemmal in their mode of termination. These relationships, if admitted, would seem to indicate a centrifugal conduction along these fibre paths of nerve impulses, presumably of motor and possibly of tonic contractile character.

LIST OF LITERATURE REFERRED TO IN THE TEXT.

(*Fuller Bibliographies will be found in Boeke's Papers, especially those of* 1911 *and* 1916-17.)

AGDUHR, E. "Morphologischer Beweis der doppelten (plurisegmentalen) motorischen Innervation der einzelnen quergestreiften Muskelfasern bei den Säugetieren," *Anatomischer Anzeiger*, 1916, **49**, 1-13.

Idem. "Ueber die plurisegmentelle Innervation der einzelnen quergestreiften Muskelfasern," *Anat. Anzeiger*, 1919, **52**, 273.

Idem. "Sympathetic Innervation of the Muscles of the Extremities: a Histo-experimental Study," *Verh. d. K. Akad. v. Wetensch. Amsterdam*, 1919.

BOEKE, J. "Die motorische Endplatte bei den höheren Vertebraten, ihre Entwickelung, Form und Zusammenhang mit der Muskelfaser." *Anat. Anzeiger*, 1910, **35**, 193.

Idem. "Beiträge zur Kenntnis der motorischen Nervenendigungen," *Internat. Monatschr. f. Anat. u. Physiol.*, 1911, **28**, 377.

Idem. "Die doppelte (motorische und sympathische) efferente Innervation der quergestreiften Muskelfasern," *Anat. Anzeiger*, 1913, **44**, 343.

Idem. "Studien zur Nervenregeneration," I, *Verh. d. K. Akad. v. Wetensch. Amsterdam*, 1916, **18**, 91.

Idem. "Studien zur Nervenregeneration," II, *Verh. d. K. Akad. v. Wetensch. Amsterdam*, 1917, **19**, No. 5, 1-71.

DE BOER, S. "Die Bedeutung der tonischen Innervation für die Funktion der quergestreiften Muskeln," *Zeitschr. f. Biol.*, 1915, **65**, 239.

BOTEZAT, E. "Fasern und Endplatten von Nerven zweiter Art an den gestreiften Muskeln der Vögel," *Anat. Anzeiger*, 1910, **35**, 396.

BOTTAZZI. *Journal of Physiol.*, 1897, **21**, 1; *Archiv f. Anat. u. Physiol.*, 1901, Phys. Abth., 377.

BREMER, L. "Ueber die Endigungen der markhaltigen und marklosen Nerven im quergestreiften Muskel," *Arch. f. mikr. Anat.*, 1882, **21**, 165.

CAVALIÉ, M. "Sur les terminaisons nerveuses motrices et sensitives dans les muscles striés chez la torpille," *Compt. rend. Soc. de Biol.*, Paris, 1901, **54**, 1279.

DOGIEL, A. S. "Methylenblautinktion der motorischen Nervenendigungen in den Muskeln der Amphibien und Reptilien," *Arch. f. mikrosk. Anat.*, 1890, **35**, 305.

DUSSER DE BARENNE, J. G. *Pflügers Arch.*, 1917, **166**, 145.

Idem. *Verh. d. K. Akad. v. Wetensch. Amsterdam*, 1919, **21**, 1231.

FLORESCO, N. "Influence de la resection du nerf sympathique cervical sur les plaques motrices et les vaisseaux du muscle," *Compt. rend. Soc. de Biol.*, Paris, 1903, **55**, 228.

GEMELLI, A. "Sur la structure des plaques motrices chez les Reptiles," *Le Névraxe*, 1905. **7**, 107.

Idem. "Nuove osservazioni sulla struttura delle placche motrici e dei fusi neuro-muscolari," *Monitore Zool. Ital.*, 1906, **17**, 90.

GERLACH, J. "Ueber die Einwirkung des Methylenblaus auf die Muskelnerven des lebenden Frosches," *Sitz. Ber. d. math. phys., Kl. d. Bayer. Akad. d. Wiss.*, 1889, **19**, 125.

GRABOWER. "Ueber Nervenendigungen in menschlichen Muskel," *Arch. f. mikr. Anat.*, 1902, **60**, 1.

KALLIUS, E. "Endigungen motorischen Nerven in der Muskulatur der Wirbeltiere," *Ergebnisse der Anat. u. Entwick.*, 1896, **6**, 26.

MOSSO, A. "Théorie de la tonicité musculaire basée sur la double innervation des muscles striés," *Arch. Ital. de Biol.*, 1904, **41**, 183.

PEKELHARING, C. A., and V. HOOGENHUYZE, C. J. C. "Die Bildung des Kreatins im Muskel beim Tonus und bei der Starre," *Hoppe-Seyler's Zeit. f. Physiol. Chemie*, 1910, **64**, 262.

PERRONCITO, A. "Sur la terminaison des nerfs dans les fibres musculaires striées," *Arch. Ital. de Biol.*, 1901, **36**, 245.

Idem. "Études ultérieures sur la terminaison des nerfs dans les muscles à fibres striées," *Arch. Ital. de Biol.*, 1902, **38**, 393.

RETZIUS, G. *Biologische Untersuchungen N. F.*, 1892, **3**.

RUFFINI, A., and APATHY, J. "Sulle fibrille nervose ultraterminali nelle piastre motrici dell' uomo," *Rivista di Patologia nervosa e mentale*, 1900, **5**, 433.

Proceedings of the Section of Neurology of the Royal Society of Medicine.

Held March 10, 1921.

Chairman—Dr. Ernest S. Reynolds.

Discussion held in connection with the Section of Ophthalmology on Ocular Palsies.

The discussion was opened by Gordon Holmes, C.M.G., M.D., and Leslie Paton, F.R.C.S.

The addresses of the openers appear in full in the *British Journal of Ophthalmology*, June, 1921, vol. v, pp. 241 and 250.

April 14, 1921.

Dr. Riddoch described certain nociceptive reflexes of the upper limb (this paper will appear in a subsequent number of *Brain*).

Dr. Brooke demonstrated a model of the brain and spinal cord.

May 12, 1921.

A paper by Prof. Marinesco was read on "A Case of Myoclonic Encephalo-myelitis of Malarial Origin." This paper appears on p. 223 of the present number.

Notices of Recent Publications.

Klinische und anatomische Beiträge zur Pathologie des Gehirns. Von Professor Dr. S. E. Henschen.

Fünfter Teil. "Ueber Aphasie, Amusie und Akalkulie," S. 216, mit 15 Tafeln.

Sechster Teil. "Ueber sensorische Aphasie," S. 231, mit 11 Tafeln.

Stockholm : Nordiska Bokhandeln. 1920.

In 1912, Professor Henschen reached his sixty-fifth year and retired from the post of Director of the Medical Clinic in Stockholm. He then devoted himself to the mass of material and observations which he had collected during his active life, and published three authoritative monographs on the auditory, the visual and the olfactory centres. Like all who have busied themselves with cerebral localization, he was led to consider the problem of aphasia, a riddle which has occupied a prominent place in medicine for nearly a hundred and twenty years. His profound knowledge of cerebral anatomy revealed to him the shaky ground on which much of the current teaching was based, and he determined to tabulate and publish in abstract every case of these disorders of speech in which there had been a sufficient anatomical examination. These two volumes form a portion of this colossal task.

The arrangement is throughout based on the same principles. The clinical manifestations are set out usually under the headings of word-deafness, word-blindness, motor aphasia, agraphia, amnesia, paraphasia and accessory symptoms. The nature and situation of the lesion is given as nearly as possible and in certain instances a drawing of the brain is to be found on one of the many tables. Then follows a short abstract of each case under the name of the observer, numbered consecutively. At the end of each section is a short general summary of the conclusions which emerge from such revision.

The first of these volumes begins with a short re-statement of the author's own personal contributions to the case-material of aphasia ; he then passes on consider amnesia and "akalkulie," of which he has collected 304 examples. As the result of this analysis he arrives at the conclusion that comprehension of music is predominantly due to the activity of the polar portion of the temporal lobe. When this centre on the left side is destroyed, that on the right can occasionally assume the lost function, but this is by no means invariable. He complains that the data for a similar examination of lesions responsible for "akalkulie" are unsatisfactory, because it is often

impossible to decide whether numbers could be written and understood or not. A + or O on the table does not give an exact expression of the facts; for the patient may be able to add but not to multiply. The author considers, however, that the mechanism for reckoning is separable from that for speech and music, and rests on a different anatomical substratum.

Fasciculus VI opens with a collection of cases of temporal and so-called transcortical aphasia, and then each group is considered separately. Henschen concludes that auditory speech is lost without recall on destruction of T.1. It cannot be replaced by the right hemisphere, which shows its inferiority to the left, exactly as is the case with F.3 in motor speech. But the right hemisphere is responsible for primitive automatic echo-speech and particularly for emotional expression.

As the result of his analysis of cases of so-called transcortical sensory aphasia, he comes to the conclusion that this form stands on a sound basis. The name "transcortical" signifies that the disturbance lies on the other side of the cortical areas for the formation of speech, and that the lesion is not situated in the so-called speech centres.

Then follows a systematic consideration of "angular," "parietal" and "occipital" aphasia, together with the combined forms.

The author comes to the final conclusion that investigation of word-deafness and word-blindness points to a strict localization of the forms of aphasia and to a close organization in the structure of the brain of the elements of speech, including the psychical functions. There is an enormous adaptive power in the brain cells which can be brought out as the result of suitable education and training; the right half of the brain is a colossal uncultivated field with innumerable and little educated cells, and this virgin ground may possibly be made to yield a full crop, although now it stands on a low level of development.

Professor Henschen promises us another volume on "Motor Aphasia," following the same lines, and all who are interested in cerebral pathology must sincerely hope that he may be spared to finish this incredible labour. For however much we may differ from the conclusions which he draws from this anatomical material, it is of the greatest importance that the recorded facts should be collected and analysed by so distinguished an anatomist. We are all anxious to know the site of the various lesions that can disturb speech, and it is often possible to see through the insufficient clinical records the main factors in the disorder. Round and round like a stage army moves the procession; the clinical appearances are identical, but each fresh group of spectators views them with new eyes and with different preconceptions. But it is often possible to recognize through the hazy reports and different classification of previous generations some salient point which indicates the identity of some past case with one under examination to-day. It is therefore of fundamental importance to know the site and nature of the anatomical lesion which produces such a condition, and we cannot be sufficiently grateful to the author of these monographs for this wonderful collection of data which can only be utilized to the full by our successors.

Anatomie du Système Nerveux. Première partie. "Les appareils nerveux de l'olfaction, de la vision, de la sensibilité générale, du goût." By Dr. CORNELIS WINKLER. 1918. Pp. 435.

In this volume an attempt has been made to describe fully certain of the organs of special sense in relation to their connections in the central nervous system. The arrangement is one to which students of neurology are now well accustomed, and it is without doubt the most useful method of presenting the anatomy of the nervous system to the clinician.

The olfactory, visual, and taste organs and the apparatus of cutaneous sensibility alone are dealt with in this volume, the apparatus of the internal ear and its connections with the central nervous system being left for a later volume. This arrangement is, no doubt, necessitated by the close relation between the vestibular nerve and the lower motor paths, but it leaves the volume somewhat incomplete as a description of the sensory organs of the human body.

The book gives a fairly full historical résumé of the progress of knowledge on the subjects dealt with, and in many places the author's own experiments and those of other neurologists of the Dutch school are given in some detail. Dogmatic statements are avoided and the reasons for the conclusions arrived at are fully stated.

The French translation has been admirably carried out by Dr. Victor Willem. The illustrations, in which for the most part the Latin terminology is employed, are numerous and clear.

The book is one which will repay serious study as a whole, and is of considerable value as a work of reference.

Einführung in die Lehre vom Bau und den Verrichtungen des Nervensystems. By the late Professor Dr. LUDWIG EDINGER. Third Edition, 1921. S. 233. Edited by Professor Dr. KURT GOLDSTEIN and Professor Dr. A. WALLENBERG.

The second edition of this book, published in 1912, has been out of print for some years, and a third edition was urgently called for. The editors have, however, contented themselves for the present with re-editing the book in its previous form, leaving to a fourth edition the alterations which have been necessitated by recent progress in our knowledge.

The present volume is a useful introduction to the anatomy and physiology of the nervous system. Its 176 illustrations and diagrams are well conceived and clearly reproduced; the text is not only brief, and to the point, but avoids to an unusual extent the error of giving dogmatic statements on unsettled points which is so common in small books on this subject. The absence of an index reduces to some extent the value of the book, and we hope this may be rectified in the next edition.

Addresses on Psycho-Analysis. By J. J. PUTNAM, M.D., Emeritus Professor of Neurology, Harvard University. With a Preface by SIGM. FREUD, M.D., LL.D. Pp. 470. London: The International Psycho-Analytical Press, 1921.

Psycho-Analysis and the War Neuroses. By Drs. S. FERENCZI (Budapest), KARL ABRAHAM (Berlin), ERNST SIMMEL (Berlin), and ERNEST JONES (London). Introduction by Prof. SIGM. FREUD (Vienna). Pp. 59. London: The International Psycho-Analytical Press, 1921.

The urgent necessity for dealing therapeutically with large numbers of patients suffering from mental illnesses in the recent War was the means of widening interest in psychological methods. The claims of the various schools of clinical psychology were investigated, and it was soon realized that Freud in his work had done much more than originate his sexual theory of the neuroses His conceptions of mental mechanisms underlying the production of symptoms in states of emotional stress were perceived by many workers to be of more than theoretical interest. Psycho-analysis has gained an established place in neurology as a therapeutic measure of value under suitable conditions and in selected cases. At the same time many consider that its greatest value, however, in dealing with psychoneuroses is not as a method of treatment, but as a means of exploring unconscious mental processes for purposes of investigation.

The extension of interest in psycho-analytical methods in different countries has led to the establishment of an International Journal of Psycho-Analysis and an International Psycho-Analytical Library, both of which are edited by Dr. Ernest Jones. The volumes under review form the first two numbers of the library to be published in English.

In number one are republished a selection of the numerous papers upon psychological subjects written by the late Dr. J. J. Putnam in the last ten years of his life. Putnam, as Freud points out in the preface, was the first American to study psycho-analysis and, as a result of testing its value in practice, became an earnest although critical exponent of it. The book forms an interesting record of his views on the psychological doctrines of Freud, as well as those of Jung and Adler, and not the least of its attractions is the charming style in which it is written.

The second volume consists of four papers on psycho-analysis in relation to war neuroses. The first three were delivered by Austrian and German psychologists at the Fifth International Psycho-Analytical Congress at Budapest in 1918, and the fourth, written by Ernest Jones, is reprinted from the *Proceedings of the Royal Society of Medicine*, London. While all the writers admit the importance of the instinct for self-preservation in the production of war neuroses, some of them claim, and the others strongly suspect, that the real basic factors on one side are sexual in origin. Realizing, however,

the inadequacy of the original sexual hunger theory as an explanation of the traumatic neuroses of peace and war, they invoke the recently developed theory of narcissism or self-love. For them, as for Freud, danger to life is an etiological factor of insufficient strength in itself to lead to severe neurotic troubles, and assert as a belief that extensive mental analysis would bring to light underlying sexual influences of primary importance. But it is by adopting an attitude of this kind, which is unsupported by convincing evidence, that the followers of Freud do much to bring into discredit his theories and methods, and to obscure what is admittedly of great value in them.

Lunacy in India. By A. W. OVERBECK-WRIGHT. Pp. 406. London: Baillière, Tindall and Cox, 1921.

The objects of this book are stated by the author to be threefold: "to summarize the condition of lunatics in India and the means available for treating them; to emphasize the importance of toxæmias as etiological factors in the production of a very large proportion of such cases . . . . and lastly, to place on record views to which a wide and varied experience of the East, spread over more than nineteen years, has given rise." Unfortunately the author has attempted to write a complete work on Mental Medicine, and has included much that can be found in ordinary text-books. But the illustrative cases are taken from his own experience in the East, and many of them contain fascinating suggestions for further investigation. His comments throw a valuable light on the customs of the people with whom he has had to deal, but they are too short, and the author's diffidence has robbed us of much interesting information. If this book comes to a further edition, we should strongly recommend that the more specific aspects of the subject should be given greater space at the expense of the more commonplace generalizations.

Diseases of the Nervous System. By SMITH ELY JELLIFFE, M.D., Ph.D., and WILLIAM A. WHITE, M.D. Third Edition, revised, rewritten and enlarged. Pp. 1,018. Illustrated with 470 Engravings and 12 Plates. Philadelphia and New York: Lea and Febiger, 1919.

This well-known text-book has been largely re-written. The first chapter is concerned with methods of examination which even include a few words on the technique of psycho-analysis. Then follow 160 pages devoted to vegetative and visceral neurology together with the endocrinopathies. Part II deals with the sensori-motor systems; affections of the cranial and peripheral nerves, lesions of the spinal cord, brain-stem, cerebellum and cerebrum are then treated systematically in consecutive chapters, and a remarkable amount of information has been gathered into a small compass. The third portion of the book is devoted to the neuroses, psycho-neuroses, and psychoses, under

the heading of "Psychical or Symbolic Systems." The diagrams and figures are well reproduced, and we are glad to see that some of the more important monographs, from which the facts have been derived, are mentioned at the bottom of the page, so that the student can follow up points in which he is interested.

Mental Hospital Manual. By JOHN MACARTHUR, M.R.C.S., L.R.C.P. Pp. 215. London: Oxford Medical Press, 1921.

This is a straightforward account, in simple language, of the duties of an assistant medical officer and of his responsibilities both legal and medical. It gives in concise and handy form the sort of advice that would be offered to a junior on beginning work in an asylum. Copies are given of the various documents used in mental work, and they are carefully explained. This little book should be of considerable help to a young man contemplating the career of an A.M.O., or about to take the post of *locum tenens* in an asylum.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I to XXIII inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

[NOVEMBER, 1921.]

BRAIN.

PART 3, VOL. 44.

STUDIES OF CEREBRAL FUNCTION IN LEARNING. No. III.—THE MOTOR AREAS.

BY K. S. LASHLEY,
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IN their first study of the electro-stimulable cortex of the dog, Fritsch and Hitzig [5] ascribed to it the function of control of voluntary movement, although they recognized that its destruction resulted in a difficulty rather than a complete loss of ability to make voluntary movements. The latter fact was largely disregarded by the investigators following them and the view that the fibres descending from the stimulable area form the chief efferent path from the cortex was adopted by Ferrier, Carville and Duret, Luciani, Bechterew, and other early workers, so that this theory has come to be accepted as one of the most firmly established facts of cerebral localization. From the first, however, certain facts, derived chiefly from studies of recovery from paralysis following lesion to the motor area, have seemed incompatible with this view. Fritsch and Hitzig emphasized the importance of other descending tracts, and Carville and Duret [4] showed that recovery from hemiplegia is not due to the vicarious functioning of the uninjured stimulable area. The belief that recovery from paralysis is only a revival of depressed reflex functions and that the power of voluntary movement is permanently abolished by complete destruction of the motor area was disproved by Rothmann [16] who observed the formation of motor habits in monkeys after destruction of the stimulable area of one hemisphere and the pyramidal and rubrospinal tracts of the opposite side.

Such experiments show that the extra-pyramidal fibres are capable of mediating complex activities of the habitual or voluntary type but

leave undecided the question whether this is their normal function or merely a vicarious function due to the destruction of the stimulable cortex. In a previous study of the rôle of various parts of the cerebrum of the white rat in the formation and retention of visual habits the writer found that the habit of discrimination of light intensities survived the total destruction of the stimulable area. Injury to an area on each occipital lobe corresponding to Brodmann's *area striata* [2] resulted in the complete loss of the visual habit, but any other third of the cortex might be destroyed without in the least disturbing the animal's ability to make the discrimination accurately. The data reported in that study seem to prove that the conditioned-reflex arcs involved in visual habits descend from approximately the same area of the cerebral cortex to which they are projected and do not traverse the cortex to descend from the stimulable areas. The visual habits have cerebral representation, but the supposed motor areas have no direct function in their performance [10].

In higher animals, especially the primates, complete paralysis of voluntary movement may follow destruction of the stimulable cortex, but this does not necessarily imply an interruption of the direct conditioned-reflex arcs. Such an explanation is to be preferred on grounds of simplicity, if contradictory facts do not appear, but disturbances of some of the mechanisms of tonic reinforcement, not in the direct conditioned-reflex path (section of the afferent nerves of a limb, cerebellar injury, &c.), may produce somewhat similar disturbances of voluntary movement and indicate an alternative explanation of the function of the stimulable areas. The demonstration that, in the rat, the stimulable area is not traversed by the conditioned-reflex arcs of visual habits seems sufficient evidence to raise the question of whether the so-called motor area is really a part of the mechanism for voluntary movement or is merely a part of the subsidiary tonic and postural mechanism.

Generalization from the rat to higher forms is complicated, however, by the probable transfer of function from the corpus striatum to the stimulable cortex, with ascent in the evolutionary scale. In the rat, destruction of the stimulable areas produces no detectable motor disturbances, but simultaneous destruction of the motor area and the corpus striatum produces a paralysis which is relatively permanent and which resembles the effect of destruction of the stimulable area in higher forms [10]. This indicates that the function of the stimulable area of primates is represented in the rat by the combined function of

the stimulable area and the corpus striatum.[1] In order that data obtained from the rat may be applicable to higher forms it is necessary, therefore, that both the stimulable cortex and the corpus striatum be considered. My earlier study eliminated only the stimulable cortex from the habit mechanisms. In the experiments reported below I have tested the effects of simultaneous destruction of the stimulable areas and the caudate portions of the corpora striata upon the rat's ability to form and retain visuo-motor habits. The data outlined above make it fairly certain that in the combined lesion we are dealing with structures analogous in function to the stimulable areas of the primates and that conclusions valid for higher forms may be drawn from the experiments.

The chief question which the following experiments were designed to answer is, then, whether or not the structures of the rat's cerebrum, injury to which produces motor disorganization, are directly concerned in the performance of habitual acts. Visuo-motor habits are best suited for such tests, since they are easily acquired and recognized. A visual area in the occipital region is clearly defined and it has been shown that cortical representation of the habit is retained after 1,400 trials of overtraining [11], so that the possibility that the habits are carried out at subcortical levels is ruled out.

The Topography of the Stimulable and Visual Areas of the Cerebrum and of the Corpus Striatum in the Rat.

The stimulable area in the rat includes the antero-dorsal third of the cerebral cortex. It extends from above the anterior margin of the hippocampus forward to the frontal pole, covering the median half of the dorsal convexity in this region. On the frontal pole it extends laterally and then caudally over the inferior orbital surface. Fig. 1 shows a composite map of the area in about twenty-five animals, with the movements elicited by bipolar stimulation. In the majority of animals the area does not extend so far caudally as in the diagram and only movements *a*, *d*, *i*, *j*, and *p* can be elicited.

The visual function seems limited to a small area on the dorsal

[1] Since the pyramidal fibres are scattered throughout the caudate nucleus it is impossible to destroy the latter alone. In earlier work [10] I found one case with extensive degeneration of the right caudate nucleus involving few of the pyramidal fibres and leaving the greater part of the stimulable area intact. This animal showed an unusually rapid and complete recovery from the motor disturbances following operation and led to the tentative conclusion that the striate nucleus and stimulable cortex have interchangeable function.

convexity of the occipital pole. Fig. 2 is a composite diagram of the lesions in nine animals in which habits of visual discrimination persisted after bilateral injury. Detailed descriptions of these animals have been given in previous papers [10, 11]. In each animal the area destroyed included about one-fourth the blackened area of the diagram. Fig. 3 is a composite diagram of the lesions in seven animals which lost the habit of visual discrimination after operation. These two series of animals show clearly the restriction of cortical function in vision to the

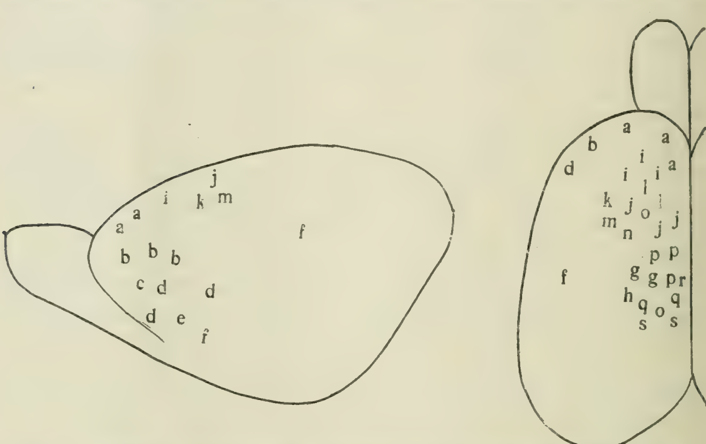


FIG. 1.—Composite diagram of the electro-stimulable points on the cerebral cortex of the rat. a = Head turned to opposite side. b = Nose retracted. c = Vibrissæ moved. d = Chewing movements, e = Tongue protruded. f = Eye closed. g = Ear adducted. h = Ear erected. i = Shoulder drawn forward. j = Fore-arm retracted. k = Elbow flexed. l =Elbow extended. m = Wrist flexed. n = Fore-arm rotated. o = Back flexed to opposite side. p = Hind leg drawn forward. q = Homolateral leg flexed, ipselateral extended. r = Ankle extended. s = Tail drawn to opposite side. All movements are contralateral to the hemisphere stimulated, except where indicated.

occipital pole and the survival of the habit after destruction of the motor area. The area included in fig. 3 corresponds roughly to Brodmann's *area striata* in other rodents [2].

The corpus striatum in the rat is very large in proportion to the volume of the cerebral cortex. Fig. 4 shows it in frontal and horizontal sections through the levels used in later diagrams. The caudate nucleus consists of masses of cells scattered among the fibres of the internal capsule and is nowhere distinct from the descending fibres. The lenticular portion is relatively free from pyramidal fibres. The

experimental lesions described below are restricted to the caudate nucleus and this is also the part whose injury produces paralytic symptoms [10, 12].

Experimental Methods.

Training.—For the study of visual habits a discrimination box of the type designed by Yerkes was used. It consists of a central compartment opening into two parallel alleys which lead by lateral passages

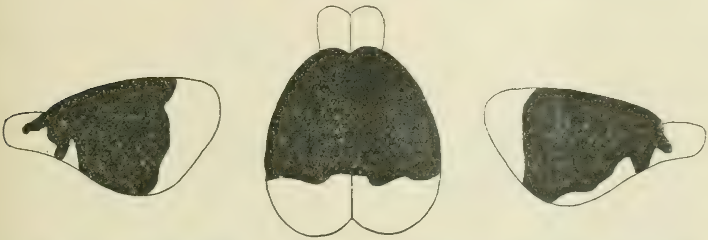


Fig. 2.—Composite diagram of the lesions in animals which retained the habit of visual discrimination after operation. The habit survived the destruction of any third of the blackened area.

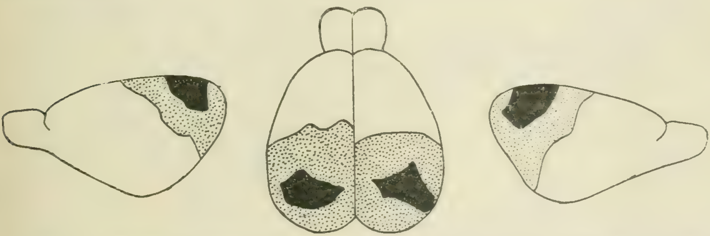


Fig. 3.—Composite diagram of the lesions in seven animals which lost the habit of visual discrimination after operation. The area which was destroyed in all is shown in solid black.

to compartments in which food is placed. At the end of each alley is a small electric bulb, 5-watt frosted "Mazda," visible from the discrimination compartment. One bulb is lighted and the other darkened in irregular alternation and the animal is trained to choose the illuminated alley, receiving food there and punishment in the darkened one. Training was continued with ten trials per day by the usual method of irregular alternation until the animals made thirty consecutive trials without entering the darkened alley. Controls were intro-

duced to assure that the reaction was to the light and not to accidental cues.

Operative methods.—Under ether anæsthesia the skull was pierced on each side of the median line at the fronto-parietal suture and the bone clipped away to make an opening, about 2 by 6 millimetres, above the caudate nucleus. Through this opening a small electric cautery, heated to redness, was plunged to a measured depth, then drawn slowly back and forth at a nearly constant depth throughout the length of the

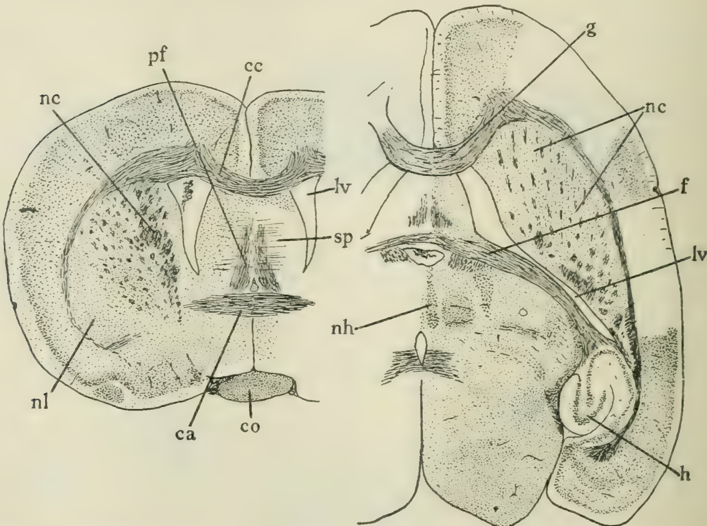


Fig. 4.—Frontal and horizontal sections through the corpus striatum at the levels of its maximum area. *c.a.* = Commissura anterior. *c.c.* = Corpus callosum. *p.f.* = Columna fornicis. *c.o.* = chiasma opticum. *f.* = fornix. *g.* = Genu corporis callosi. *h.* = Lobus hippocampus. *l.v.* = Ventriculus lateralis. *n.c.* = Nucleus caudatus. *n.h.* = Nucleus habenulæ. *n.l.* = Nucleus lentiformis. *sp.* = Septum pellucidum.

caudate nucleus. A fine scalpel was next passed from the posterior margin of the skull opening to the base of the olfactory bulb and drawn back and forth with its point in contact with the floor of the cranial cavity, so as to sever the frontal pole from the remaining cortex. In the majority of cases this method resulted in the complete destruction of the stimulable areas and in extensive lesions to both caudate nuclei. When hæmorrhage was stopped, the scalp was closed with interrupted s ures and covered with a cotton-collodion dressing.

Retention tests.—Animals trained before operation were tested in the problem box as soon as their general condition permitted; usually twenty-four hours after operation. In these tests for retention of the habit the same method was employed as in training, except that no punishment was given for errors during the first fifty trials; that is, the retention test is essentially a retraining in the habit and evidence for retention consists in the reappearance of accurate discrimination in significantly less time than was required for the original learning.

When a habit is lost after operation the question of post-operative shock must be considered. In earlier papers I have discussed methods of distinguishing between loss due to shock and that due to destruction of a functional area. In the present experiments positive evidence for retention following operation was obtained in every case, so that the question of shock does not enter, save as an explanation of the few errors made in the trials immediately following operation.

Reconstruction of lesions.—After completion of the retention tests the brains were removed and serial frontal sections stained with iron hæmatoxylin were prepared. Camera outlines of sections at intervals of one half millimetre were made and the extent of the lesions indicated on them. The positions of the sections were determined from internal structures and indicated on diagrams of these structures projected to the surface of the brain. The dimensions of the lesions were then transferred with proportional dividers to the diagrams, the points so determined connected by lines and the areas inked in. The reconstructions were finally verified by reference to the sections.

Only easily recognizable lesions, absorption or complete degeneration of the cortex and subcortical nuclei were recorded. The diagrams, therefore, represent the minimal destruction produced by the operations. In all cases, probably, larger areas than those shown were destroyed, but, since the experiments deal with the non-function of given areas and do not seek to localize functions accurately, this fact does not invalidate the results.

Graphic and tabular presentation of results.—In the preparation of the figures a uniform system has been followed. Diagrams of the superficial lesions made from camera outlines of an average brain are given at the top of the figure. Below at the left is a diagram of a horizontal section through the cerebrum at the level of the maximum area of the caudate nucleus. Practically all of the ganglion cells of that nucleus are included within the dotted line, the posterior horn being made up almost altogether of the fibres of the internal capsule.

At the right, below, is a camera outline of a frontal section through the cerebrum at the level of the anterior commissure (except in fig. 12 where the section lies at the level of the anterior horns of the lateral ventricles). In these outlines complete replacement of nervous structure by scar tissue or amorphous material is indicated in solid black; marked degeneration with some nerve elements intact is shown by stippling.

In the records of training and retention the "number of trials required for learning" represents the number of times that the animal traversed the problem box from the starting compartment to the food, before thirty consecutive trials were made without entrance into the dark alley. Ten trials were given each day in training and retention tests except where indicated. The records of these trials are given as horizontal rows of figures, each number representing the number of trials, in the ten of that day's practice, in which errors were made. Thus in the record of No. 17, 4 : 2 : 1 : 2, &c., means that on the first day four of ten trials contained errors, on the second two of ten, &c. Five errors in ten trials shows no discrimination. When no error is made for thirty trials, it is rare that errors occur in later trials unless the animal is disturbed by being roughly handled or by some new elements in the situation, such as those introduced by scrubbing the problem box.

The Formation of Habits after Destruction of the Stimulable Areas and the Caudate Nuclei.

The formation of maze, latch-box, and visual habits after destruction of the stimulable area in the rat has been reported in earlier papers [10, 12]. In the present study I have attempted to train animals after destruction of both the stimulable areas and the caudate nuclei. Only three animals have survived the operation long enough to give evidence of learning and they have formed only simple habits. The individual records of these animals follow.

No. 17.[1] Adult male.—The cautery was plunged into each caudate nucleus and the frontal pole of each hemisphere was incised. Training for visual discrimination was begun eight days after operation. For several days he spent most of the time climbing to the top of his cage and falling back to the floor. When placed in the problem box he promptly climbed out and repeated this more than two hundred times in spite of severe punishment. He

[1] For convenience in reference these animals are numbered consecutively with others of the visual series which have been reported earlier [10, 11].

was then placed in the left-hand feeding compartment in contact with the food, and began to eat immediately. By the process of removing him a few inches farther from the food at each trial, he was trained to find his way through the training box from the starting compartment to the food. He was then given regular training in visual discrimination for seven days. In this time he showed no evidence of visual discrimination, but formed a position habit toward the left alley, which was fixed in a normal learning curve, with errors (turns to the right) as follow, in successive tens of trials :—

4 : 2 : 1 : 2 : 1 : 0 : 0.

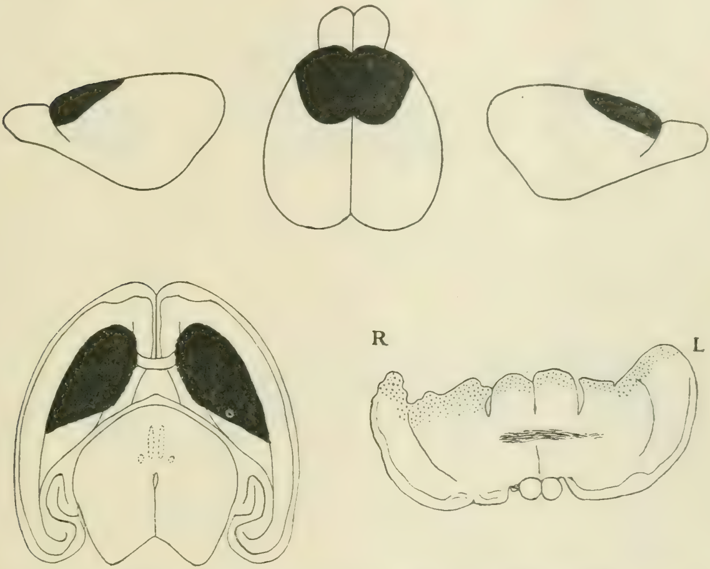


FIG. 5.—Extent of lesions in No. 17. In this and the following diagrams a uniform arrangement has been adopted. Above, the lesions to the cortex are shown in lateral and dorsal aspects. Below, at the left, is a diagram of the horizontal extent of the lesions to the caudate nuclei, in this case embracing the entire extent of both nuclei. At the right is a camera outline of a frontal section at the level of the anterior commissure, showing the depth of the lesion. In these outlines degenerated areas are stippled and scar tissue, which does not appear in this one, is shown in solid black. The outlines of the frontal sections are reversed, the right side appearing on the left. The diagrams are correctly orientated.

Ten days after operation he developed a marked spasticity and died on the eleventh day. Autopsy showed the wound infected, with extensive abscesses in both hemispheres, extending into the lateral ventricles.

Extent of lesions (fig. 5).—Right hemisphere: The entire cortex of the dorsal surface of the hemisphere, from the frontal pole to the level of the

pillars of the fornix, is replaced by a cyst. The dorsal half of the striate nucleus, including all of the caudate nucleus, is absorbed. The lenticular nucleus is uninjured. Left hemisphere: The lesion is identical with that on the right.

No. 18. Small adult male.—The frontal lobes were transected and the caudate nuclei cauterized. For eleven days the animal showed marked disturbances of movement, rotating to the right, so that he was unable to eat without assistance. By the twelfth day this trouble had partially cleared up, so that he could walk in a straight line—although he still tended to rotate

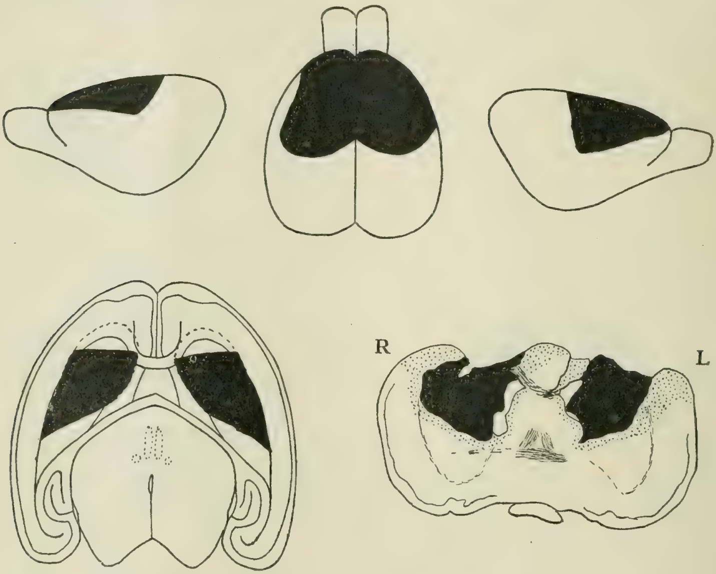


FIG. 6.—Extent of the lesions in No. 18. Arranged as in fig. 5.

when eating. Training in visual discrimination was begun on the fourteenth day after operation. At first he had great difficulty in making the turns in the training box and in finding food, since the operation rendered him anosmic. In five days (fifty trials) he formed the somæsthetic-motor habits of the training box, so that he could go directly from the starting compartment to the food, correcting the error and orientating promptly when he failed to find food in the darkened alley. He still had some difficulty in recognizing food, and would eat shavings and fæces in the neighbourhood of the food dish, but not elsewhere. He died twenty-three days after operation.

Extent of lesions (fig. 6).—Right hemisphere. The lesion to the cortex

extends as a diagonal incision from the level of the thalamico-mamillary tract to the frontal pole, destroying all of the cortex of the dorsal convexity and extending over the superior part of the orbital surface. The caudate nucleus is completely destroyed from the level of the anterior commissure to its posterior margin. The thalamus is invaded at the level of the anterior and lateral nuclei. Left hemisphere: The lesion to the cortex is similar to that on the right, but does not extend so far lateral. The posterior part of the caudate nucleus is destroyed, as on the right. The anterior and lateral thalamic nuclei are destroyed.

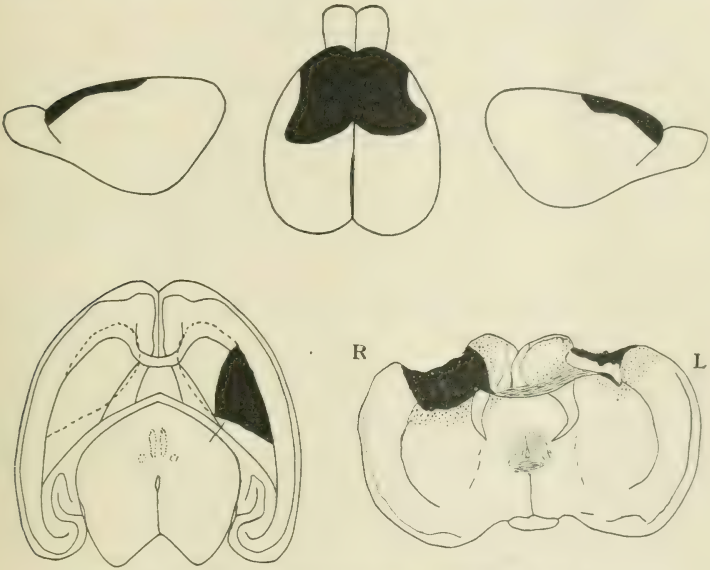


FIG. 7.--Extent of the lesions in No. 19 Arranged as in fig. 5.

No. 19. Large adult male.—The frontal lobes were transected and the caudate nuclei cauterized. Training in brightness discrimination was begun ten days after operation. The habit was formed in 130 trials with errors in successive tens of trials distributed as follows:—

6 : 6 : 2 : 5 : 5 : 2 : 1 : 3 : 1 : 1 : 0 : 1 : 1 : 0 : 0 : 0.

Extent of lesions (fig. 7).—The dorsal convexity of both hemispheres was destroyed from the level of the thalamus to the frontal pole. The orbital surfaces and olfactory tracts were not injured. The lateral portion of the right caudate nucleus was destroyed from the level of the knee of the corpus callosum to its posterior limit. The left nucleus was uninjured.

The data from these animals, in conjunction with that presented in earlier papers, establishes the fact that the rat may form somæsthetic motor habits in the absence of the stimulable cortex and of the caudate nuclei. I have shown that destruction of the stimulable area alone does not lead to an appreciable reduction in the rate of formation of visual habits, and that a difficult latch box, the "double platform box," may be learned at normal rate after complete destruction of the stimulable areas and severe injury to one or both caudate nuclei [10]. Of the three animals reported in the present study, No. 19 alone survived long enough to allow of acquirement of the visual habit and in this animal the left caudate nucleus and part of the right were uninjured. In the other two, however, the stimulable cortex was destroyed and the caudate nuclei were either destroyed, as in No. 17, or severed from their thalamic connections, as in No. 18. These animals survived only a short time, but both gave unmistakable evidence of the formation of simple somæsthetic-motor habits. From the data on the retention of visual habits after the same operation, presented in the following section, it is probable that the animals would have formed the visual habit if training could have been continued.

The problem of the relation of the extent of cerebral destruction to the complexity of the habits which may be formed must be reserved for later discussion when more adequate experimental data are available. The interest of the present study is chiefly in the question of whether or not habitual "voluntary" movements are mediated by the stimulable areas. These experiments show clearly that neither the stimulable area nor the caudate nuclei are necessary for the acquirement of such movements. This is in agreement with the results of Rothmann [16] with monkeys and justifies the conclusion that the so-called motor areas are not essential to the acquirement of voluntary activities.

This at once suggests the more important question of whether under normal conditions the motor areas are directly functional in the performance of habitual reactions. This problem has been attacked by destroying the structures after a complex habit had been acquired and testing for retention of the habit after the operation.

The Retention of Visuo-motor Habits after Destruction of the Stimulable Areas and the Caudate Nuclei.

For tests of retention after operation the animals were trained in the discrimination box until they made thirty consecutive trials without an error. When this record was attained the problem was considered

learned, but in some cases training was continued for from 30 to 60 additional trials, as it was not always convenient to operate earlier. Since I have found that over-training up to 1,400 trials does not influence cortical localization, this additional training is not objectionable. When the problem was learned the animals were subjected to operation and their retention of the habit was tested as soon as their physical condition permitted.

In training ten trials were given daily. When the animal entered the darkened alley he was punished and the trial was recorded as an error, although he was allowed to turn back and pass through the illuminated alley to the food. In the retention tests the same method was used except that the animals were not punished. In the records given below the number of errors made in each successive ten trials of training and of the retention tests is given. Five errors are to be expected from chance if the animal is not discriminating. Fewer than four errors in ten trials suggest discrimination, and no error in thirty consecutive trials usually means that the animal will discriminate accurately thereafter. The individual records of the animals follow.

No. 20. Large male.—Trained in visual discrimination. Sixty trials were required for learning, with errors in successive tens of trials distributed as follows :—

7 : 6 : 2 : 5 : 3 : 2 : 0 : 0 : 0 : 0 : 0 : 0.

The frontal lobes were transected and the caudate nuclei cauterized. The animal was first tested three days after the operation. He was slow and spastic, but made no error in twenty consecutive trials. For the next three days he was in an excited state and showed great fear, either refusing to run or dashing through the training box at random and paying no attention to the food. On the eighth day he was again normal in behaviour and gave clear evidence of discrimination.

Post-operative retention tests ; errors in successive tens of trials on consecutive days after operation :—

0 : 0 : (frightened, 6 : 4 : 3) 0 : 1 : 0 : 1 : 0 : 0.

Extent of lesions (fig. 8).—Right hemisphere : the lesion includes all of the cortex of the dorsal convexity from the level of the thalamus to the base of the olfactory bulb. It extends over the orbital surface at the level of the caudate nucleus where the cautery passed through the external capsule. The caudate nucleus is destroyed from the level of the anterior commissure to its posterior limit. There is some injury to the anterior thalamic nuclei. Left hemisphere : the lesion to the cortex is similar to that on the right, but does not extend on the orbital surface. The injury to the caudate and thalamic nuclei is similar to that on the right.

This animal showed unmistakable evidence of retention after

destruction of almost the entire stimulable cortex and of the posterior two-thirds of both caudate nuclei.

No. 21. Small female about 90 days old.—Trained in visual discrimination. Ninety trials were required for learning, with errors in successive tens of trials distributed as follows :—

8 : 5 : 5 : 3 : 2 : 3 : 0 : 4 : 1 : 0 : 0 : 0 : 1 : 1 : 0 : 0 : 0.

The frontal lobes were transected and the caudate nuclei cauterized. For the first week after operation the animal was unable to walk except in 6-inch circles and could not make her way through the discrimination box. By the

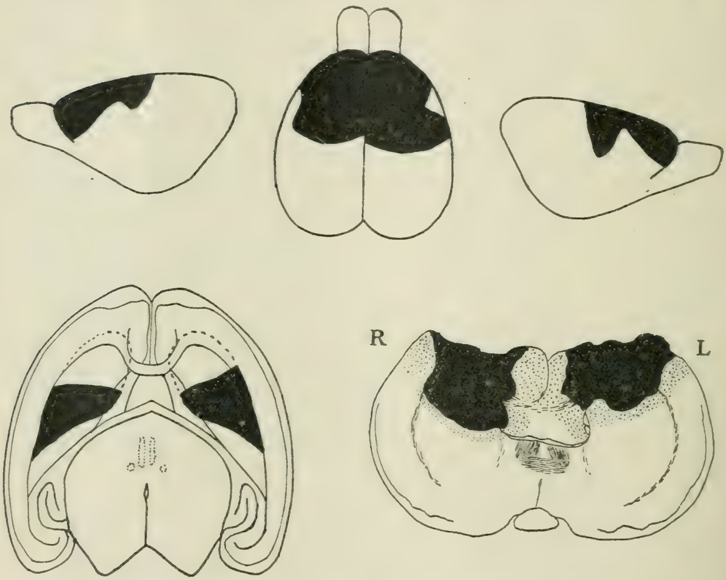


FIG. 8.—Extent of the lesions in No. 20. Arranged as in fig. 5.

ninth day after operation the motor disturbance had cleared up so that she could walk without rotation, although she still rotated when attempting to change direction and could not turn her body to the left. Retention tests were begun at this time and the animal at once gave certain evidence of discrimination. The tests were continued for twelve days. Throughout this time she was unable to turn to the left but compensated by rotating 270 degrees to the right wherever the problem box demanded a turn of 90

degrees to the left. Fig. 14 shows some of her simpler paths in traversing the problem box.

Post-operative retention tests; errors in successive tens of trials on consecutive days.:—

1 : 1 : 4 : 3 : 0 : 1 : 0 : 1 : 1 : 0 : 0.

Extent of lesions (fig. 9.)—All of the cortex of both hemispheres from the level of the thalamus to the base of the olfactory bulb is destroyed. The right caudate nucleus is completely destroyed except for a small antero-medial region laterad to the forceps of the corpus callosum. The left caudate nucleus

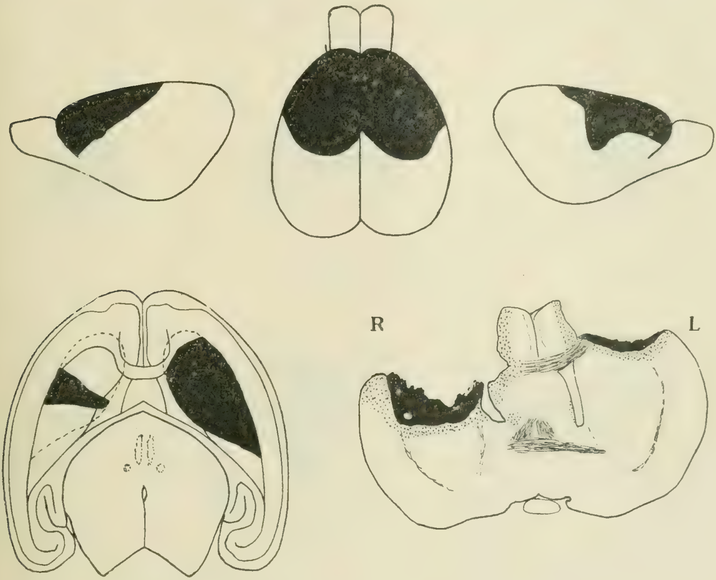


FIG. 9.—Extent of the lesions in No. 21. Arranged as in fig. 5.

has a superficial transverse lesion at the level of the anterior commissure. The right anterior thalamic nucleus is invaded.

After destruction of the stimulable cortex and severe injury to both caudate nuclei, resulting in marked motor disorganization, this animal gave certain evidence of retention of the habit of visual discrimination.

No. 22. Large male.—Trained in visual discrimination. Eighty trials

were required for learning, with errors in successive tens of trials distributed as follows :—

7 : 7 : 4 : 2 : 1 : 1 : 0 : 1 : 0 : 0 : 0 : 0 : 0.

The frontal lobes were transected and the caudate nuclei cauterized. The animal was stuporous for the first day after operation, but on the second day was in fair condition and reacted promptly to the problem box, although tending to rotate to the left. On this day he made four errors in twenty trials, but the errors were obviously due to failure to compensate for the motor difficulty and his behaviour in the discrimination compartment clearly indicated discrimination. On the following and later days he made no errors.

Post-operative retention tests ; errors in successive tens of trials :—

2 : 2 : 0 : 0 : 0 : 0.

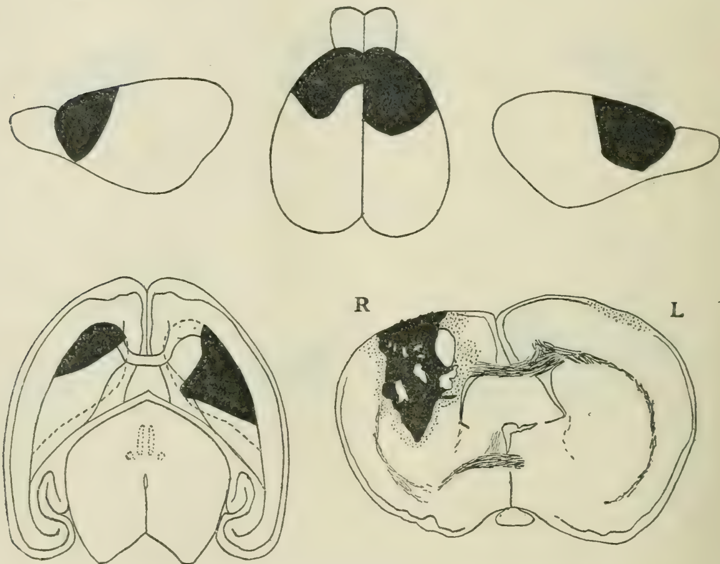


FIG. 10.—Extent of the lesions in No. 22. Arranged as in fig. 5.

Extent of lesions (fig. 10).—Right hemisphere. The lesion extends from the level of the thalamus to the base of the olfactory bulb, separating all of the cortex above this plane from the underlying structures. The cortex of the orbital surface was destroyed by the cautery. The lateral portion of the caudate nucleus is destroyed by a lesion which embraces the full width of the nucleus at the level of the anterior commissure and grows narrower cephalad and caudad to this level. Left hemisphere : The incision extends

from the level of the anterior commissure to the base of the olfactory bulb, destroying all of the cortex above this plane. All of the caudate nucleus in front of the knee of the corpus callosum is degenerated; the posterior part is uninjured.

This rat retained the visuo-motor habit after extensive but incomplete lesions to the stimulable cortex and caudate nuclei.

No. 23. Small adult male.—Trained in visual discrimination. Forty trials were required for learning, with errors in successive tens of trials distributed as follows:—

7 : 5 : 2 : 4 : 0 : 0 : 0 . 0 : 0 : 0 : 0 : 2 : 0 : 0 : 0.

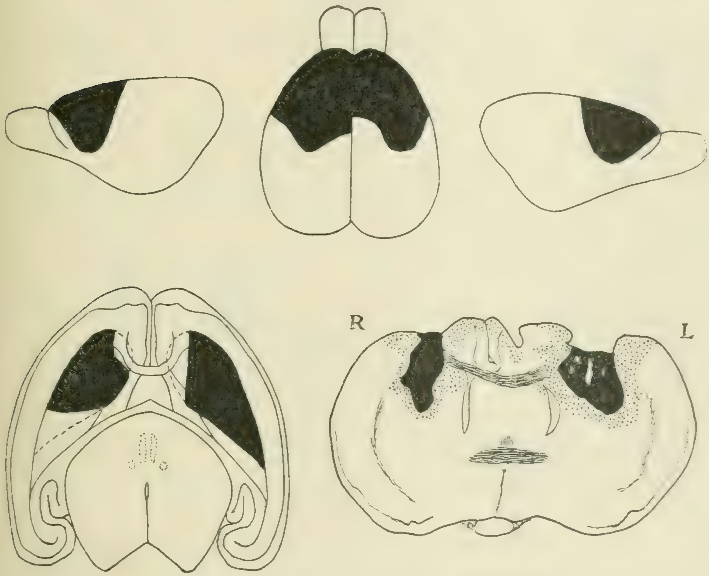


FIG. 11.—Extent of the lesions in No. 23. Arranged as in fig. 5.

The frontal lobes were transected and the caudate nuclei cauterized. For three days after operation the animal's eyes were closed and he could not be tested. On the fourth day he was in good condition and gave clear evidence of discrimination.

Post-operative retention tests; errors in successive tens of trials:—
2 : 1 : 0 : 0 : 0.

Extent of lesions (fig. 11).—Right hemisphere: The lesion to the

cortex begins at the level of the thalamus as a narrow band and broadens cephalad to include all of the cortex of the frontal pole. The lesion to the corpus striatum extends from the caudal limit of the nucleus throughout its length. All of the lateral half of the nucleus is destroyed, but the lesion probably did not involve the median part in front of the anterior commissure. The depth of the lesion is the same throughout its length, involving a little more than the dorsal third of the corpus striatum. Left hemisphere: The lesion to the cortex is practically identical with that on the right. The greater part of the caudate nucleus is destroyed, only the extreme poles remaining uninjured.

This animal showed perfect retention to the visuo-motor habit after nearly complete destruction of the motor areas.

No. 24. Small adult male.—Trained in visual discrimination. One hundred trials were required for learning with errors in successive tens of trials distributed as follows:—

4 : 5 : 3 : 5 : 1 : 3 : 4 : 0 : 2 : 2 : 0 : 0 : 0 : 0.

The frontal pole was transected and the caudate nuclei cauterized. The animal was stuporous for forty-eight hours after operation, then was tested for discrimination. He reacted slowly, but found his way through the problem box and gave some indication of discrimination, although he made frequent errors. On the fourth day of the tests he began to discriminate accurately and made no further errors.

Post-operative retention tests; errors in successive tens of trials:—

3 : 5 : 3 : 0 : 0 : 0 : 0.

The fact that the habit was reacquired in thirty trials, in contrast to the hundred of original training, shows retention rather than relearning.

Extent of lesions (fig. 12).—Right hemisphere. The lesion to the cortex begins at the level of the anterior commissure and includes all of the dorsal convexity of the frontal pole except a narrow median band. The hind leg and part of the fore leg region remain intact. All of the caudate nucleus in front of the anterior commissure is destroyed; its posterior part is uninjured. Left hemisphere: The lesion is almost identical with that on the right.

After extensive injuries to the stimulable area and destruction of the anterior halves of both caudate nuclei this animal gave evidence of retention.

No. 25. Small adult female.—Trained in visual discrimination. One hundred and ten trials were required for learning, with errors in successive tens of trials distributed as follows:—

4 · 5 · 4 : 5 : 2 : 3 : 5 : 3 : 1 : 1 : 1 : 0 : 0 : 0 : 0 : 0 : 0.

The frontal pole was transected and the caudate nuclei cauterized. For three days after operation the animal was unable to walk except in a very narrow circle to the right and could not get through the problem box. On the

fourth day she still rotated but was able to find her way through the box by keeping near the partitions, and made only one error in ten trials. On the following day she developed pronounced fear reactions and climbed out of the training box more than one hundred times. She was finally induced to make ten trials, but made six errors. Training was discontinued for five days. When she was next tested the fear had disappeared and she showed perfect discrimination.

Post-operative retention tests ; errors in successive tens of trials :—
1 : 6 : 0 : 1 : 0 : 0 : 0.

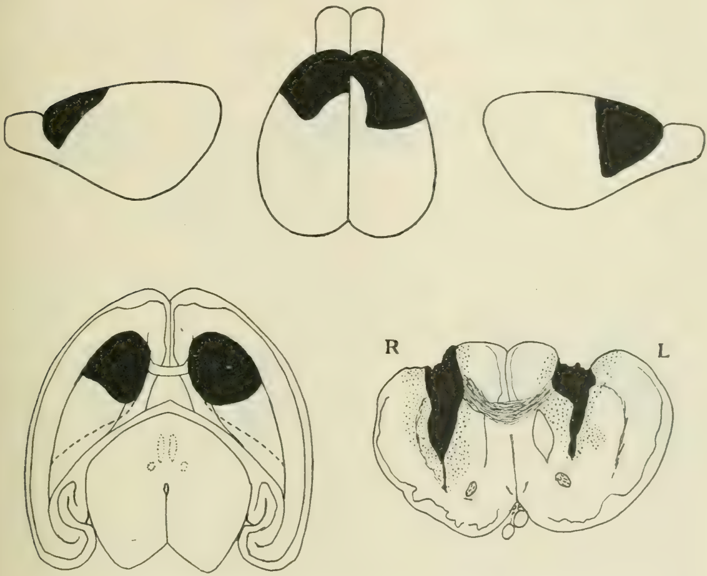


FIG. 12.—Extent of the lesions in No. 24. Arranged as in fig. 5.

Extent of lesions (fig. 13).—Right hemisphere : The lesion to the cortex begins at the level of the commissura habenulæ and extends to the base of the olfactory bulb, including all of the motor cortex except a part of the hind leg area. The cautery passed throughout the length of the caudate nucleus and the entire nucleus is degenerated. Left hemisphere : The lesion to the cortex is similar to that on the right, but misses the antero-lateral surface of the frontal pole, and does not extend so far over the orbital surface. The caudate nucleus is entirely degenerated.

Practically the entire stimulable cortex and both caudate nuclei

were destroyed in this animal. There was clear evidence of retention following the operation.

This group of animals is not selected, but comprises all that survived the operation long enough to allow of retention tests. Every one of them gave evidence of retention early in the tests. Table I gives the number of trials and the number of errors, in learning and and in the post-operative retention tests. The average number of trials required for learning is eighty. Only 33·3 trials on the average

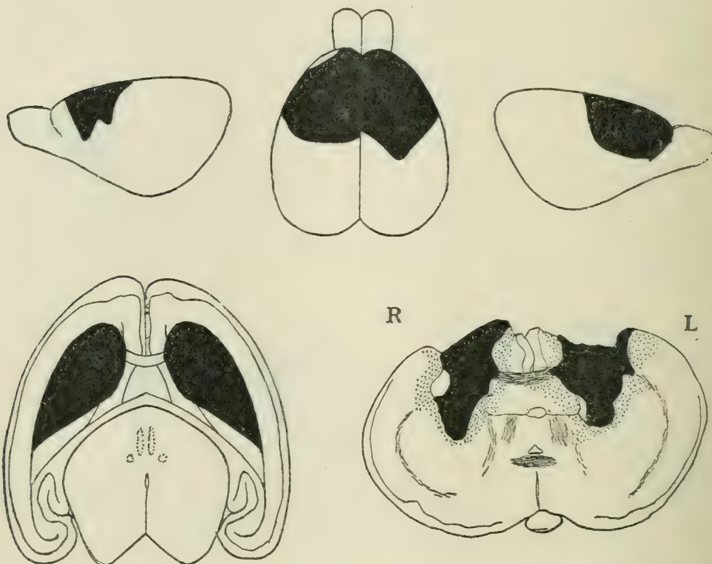


FIG. 13.—Extent of the lesions in No. 25. Arranged as in fig. 5.

were required after operation to reach the same degree of accuracy in discrimination. Errors were made in an average of 26·6 trials in learning and in only 6·3 trials in the retention tests. These figures include errors obviously due to motor disturbance and fright following operation. Thus, No. 21 was never able to turn directly to the left and all her errors seemed due to failure to compensate for the motor difficulty; No. 24 was slow and stuporous during the time when errors were made; six of the eight errors made by No. 25 were made

on a day when the animal was too disturbed to eat. If allowance is made for such errors, retention in every case was practically perfect.

TABLE I.—NUMBERS OF TRIALS AND OF ERRORS PRECEDING THE FIRST THIRTY CONSECUTIVE TRIALS WITHOUT ERROR IN LEARNING AND POST-OPERATIVE RETENTION TESTS FOR ANIMALS OPERATED AFTER TRAINING.

	Training		Retention tests	
	Trials	Errors	Trials	Errors
No. 20	60	25	0	0
No. 21	90	31	90	12
No. 22	80	23	20	4
No. 23	40	18	20	3
No. 24	100	29	30	11
No. 25	110	34	40	8
Average	80	26·6	33·3	6·3

The small operative field presented by the rat's brain and the necessity for avoiding hæmorrhage on the floor of the cranial cavity or injury to the optic nuclei of the thalamus make the complete destruction of the caudate nuclei difficult. It was accomplished only in No. 25. In this animal also the entire motor area, except the posterior part of the hind leg region, was destroyed. The animal showed motor and emotional disturbance, but gave certain evidence of retention of the habit. In No. 20 the posterior halves of both nuclei were destroyed ; in No. 24, the anterior half ; in the others the lesions were irregular, but even more extensive. The lesions to the cortex in all cases included the neck, fore-leg, and the greater part of the hind-leg regions.

Earlier experiments [10] had shown that no part of the stimulable cortex is necessary for the performance of the visual habit. The alternative explanations of this fact were : (1) that the striate nuclei function either vicariously for the stimulable cortex when the latter is destroyed, or normally as the source of efferent impulses from the cerebrum ; or (2) that the supposedly motor regions of the cerebrum do not lie in the direct path of conditioned-reflex arcs and are of only secondary importance in the performance of voluntary movements.

The foregoing results seem to prove that the second hypothesis is correct. In the absence of the stimulable cortex, injury to the caudate nuclei does not affect retention of the visual habit and perfect discriminations may appear, even after complete destruction of both nuclei. It seems clear, therefore, that neither the stimulable cortex nor the caudate nuclei are directly functional in the performance of the visuo-motor habit.

The experiments have not dealt with the lenticular nucleus and the possibility that this forms a link in the conditioned-reflex arc remains. The data previously presented on the effects of injuries to the striate nuclei [10] indicate that the caudate nucleus and the stimulable cortex together in the rat are equivalent in function to the stimulable cortex of higher forms, since their combined destruction results in relatively irrecoverable paretic symptoms. On embryological grounds there is less evidence for assuming homologous function between the lenticular nucleus and the stimulable cortex than between the latter and the caudate nucleus. In some of the earlier experiments of this series, Nos. 7 and 8 [10], the cerebral cortex and underlying association fibres were almost completely transected at the level of the anterior thalamic nuclei; in the present experiments the greater part of the internal capsule has been destroyed without abolishing visual discrimination. The cortical relations of the lenticular nucleus are not well established, but any occipito-lenticular fibres which may exist were almost certainly destroyed by these operations. It seems very improbable, therefore, that the lenticular nucleus has any more important place in the conditioned-reflex arc than has the caudate nucleus.

The evidence presented in this series of papers all points to the conclusion that in the rat the area striata on the dorsal convexity of the occipital pole is the only cerebral structure which functions in the habit of discrimination of light intensities. The habit may be formed or retained after the destruction of any other part of the cerebrum (with the possible exception of the ectorhinal and inferior temporal regions, which have not been explored but are almost certainly not functional in this habit). This means that the conditioned-reflex arcs which mediate the habit must pass to and from the cortex by way of the occipito-thalamic fibres, that the reintegrations occur within the limits of the visual area, and that long transcortical association fibres do not function in the formation or retention of the habit.

Are the conditions similar for other types of habit? There are no significant data for more complex habits. The poor vision of the rat makes the formation of complex visual habits so slow that I have not been able to carry out similar experiments with them. I have not yet located an auditory area. Certain types of latch-box habits are abolished by injury to the stimulable area [12]. The effective lesions here do not correspond in extent to the stimulable area, but only to the excitable region for the neck and fore limbs, which are used no more than the trunk and hind limbs in the manipulation of the latch. It is

probable that the loss in this case is due to the fact that the frontal region is an important somæsthetic projection area as well as electro-stimulable. The one case for which there is clear evidence is the group of somæsthetic habits of the discrimination box which determine general orientation, finding of food, responses to doors, prompt correction of errors, &c. These survive the destruction of any given third of the cortex [10] and of the subcortical cerebral nuclei. It is possible that they are formed at levels below the cerebrum; it is certain that they are not dependent upon the so-called motor structures.

There is some evidence, to be reported later, that the habit of the "double platform box" [10] is disturbed but not completely abolished by destruction of either the frontal or the occipital regions. This suggests that more complex habits involving diverse sense organs may demand co-ordination of distant portions of the cerebrum and that the visuo-motor habit dealt with in this study is too simple to give a typical picture of cerebral function. It does not, however, indicate any greater importance of the pyramidal tracts in complex habits, and we may conclude that the existing evidence for the rat all opposes the direct participation of the "motor areas" in the performance of any habit.

The Function of the Stimulable Area and the Caudate Nucleus in the Rat.

The evidence that the stimulable cortex in the rat has no direct function in the performance of habitual motor adjustments seems conclusive. What then is the function of this portion of the cortex? Data for a final answer to the question are not available, but some suggestions are given by the motor disturbances following unilateral lesion to the stimulable area and the caudate nucleus.

Immediately following combined unilateral destruction of the caudate nucleus and the stimulable cortex the animal shows a very pronounced rotation toward the injured side. Left undisturbed, he assumes a normal position, but immediately upon stimulation he bends sharply toward the side of the injury and walks in a circle, sometimes so narrow that the head is brought across the hind quarters or trips up the hind feet. The legs of the side opposite the injury are hyper-extended and spastic (fig. 15*c*). This condition may persist for several weeks, but usually clears up within a few days to such an extent that, when the animal is placed in a large open space and allowed to become orientated, he is able to walk in a straight line. But the tendency to

rotate still appears in many situations. When the animal is placed on the floor he turns around several times before taking a direct course. The necessity for avoiding an obstacle frequently results in several complete rotations. The tendency to rotate becomes more pronounced in narrow quarters and in the problem box the animals have a good deal

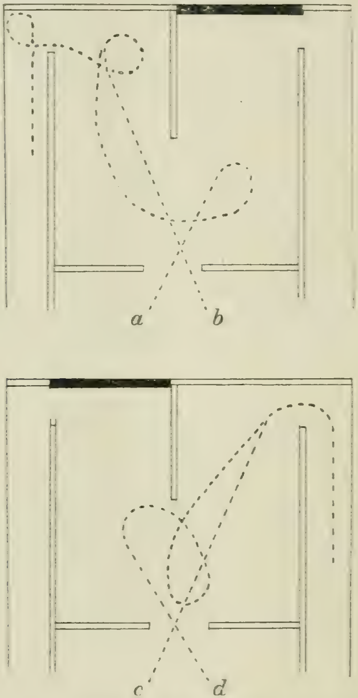


FIG. 14.—Tracings of paths followed by No. 21 in the discrimination box. The animal was unable to turn to the left but compensated for this motor difficulty by rotation to the right, as shown. The discrimination was in each case correct. The path varies with the alley illuminated and with the initial orientation.

of difficulty in making the necessary turns. Fig. 14 shows tracings of the path of animal No. 21 at a time when she was making no errors of discrimination. She was able to walk in a fairly straight line, but any need for change of direction resulted in rotation toward the right. (Only the right caudate nucleus was injured in this animal.) Her path

was usually more complicated than those shown, the turns involving not one but several complete rotations.

In feeding and scratching the animals show similar difficulty in

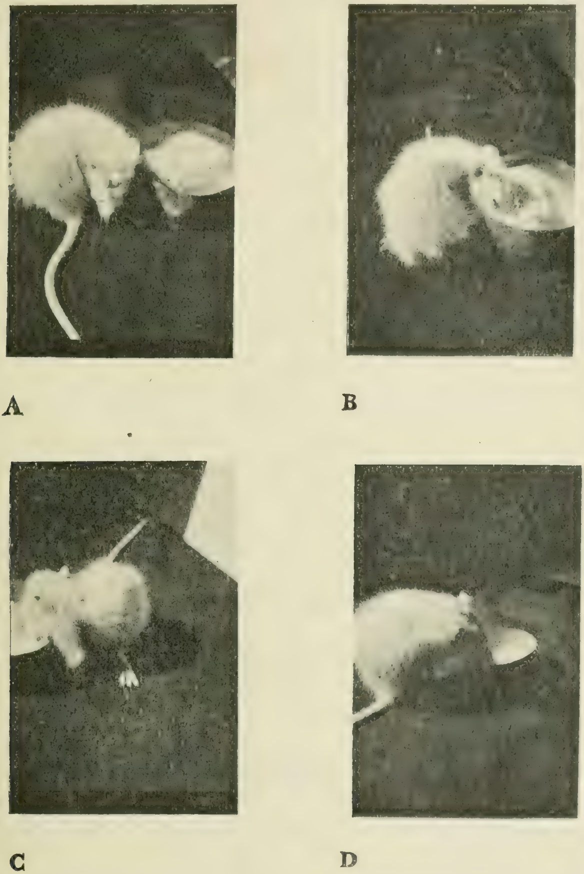


Fig. 15.—The behaviour in feeding of an animal with total destruction of the right stimulable area and caudate nucleus. For explanation see text.

adjustment. Fig. 15 shows various positions assumed by an animal with complete right unilateral destruction of the stimulable cortex and

caudate nucleus, three weeks after operation. She approached the dish, walking in a rather wide circle, but as soon as her mouth came in contact with the food, which stimulates the normal rat to squat and assume a definite feeding position, she turned sharply to the right, the position shown at A. She then rotated several times to the right until by chance her nose was braced against the side of the dish (B). A similar position with hyperextension of the left legs is shown at C. Once such a feeding position has been attained it may be held for some time and a gradual relaxation of the strained posture of the body then appears. Thus, at D, the animal was held in contact with the food for about thirty seconds, then gradually released. She then maintained the normal feeding position for nearly a minute, turning only gradually to the right, as successive bites were taken from the edge of the cube of bread, and finally becoming overbalanced and falling.

In all the activities of such animals, the greatest disturbance of co-ordination appears when some new adjustment to the environment is necessary. Initiation of locomotion, change in direction of locomotion, assumption of a sitting posture, stretching forward for food, scratching, &c., are attended by sudden rotations as though the difficulty were chiefly in gaining a new posture or in initiating a new activity. Yet the activities are initiated and the disturbances are compensated for in ways that indicate that the difficulty is not at the level of the most complex integrations. Thus, in 1917, Franz and the writer reported the case of an animal with complete destruction of the motor cortex and degeneration of one striate nucleus, which learned the "simple maze." The maze used had essentially the same ground plan as the problem box shown in fig. 14. The animal was trained to go constantly to the left. He rotated and fell constantly to the right and at first could not make the turn to the left. He finally learned to grasp the end of the partition with his left hind foot and, with this as a pivot, to swing his body to the left and so enter the food compartment. Fig. 14 shows a more common type of adjustment where the drive toward the goal is maintained in spite of the motor difficulty.

Such adaptations seem to prove that the interference is not with the more complex integrations of voluntary reactions. The compensations rather suggest those described by Luciani [13] for dogs after cerebellar injury, where the tendency to rotate is compensated for by walking with the body curved, and where the lesion admittedly interferes only with reflex tonic control.

The recent discussions of postural reflexes by Sherrington [17] and

Langelaan [9] and of their relation to decerebrate rigidity [Wilson, 19], suggest a possible explanation for the results of destruction of the cerebral motor structures in the rat. The initiation of almost any new activity demands a modification of postural tone, the assumption of a different pattern of tonic contractions, which may then persist almost unaltered for some time during the performance of finer manipulative movements. The behaviour of the operated rats suggests strongly that their chief difficulty is in taking a new posture. Once an activity has been initiated and maintained for a moment even artificially, as when the animal is held in contact with the food, it may persist for a relatively long time until some stimulus to a new posture occurs. The disturbances seem most pronounced in the attempts to perform those activities which require the most complex and unusual postural co-ordinations : scratching and feeding. The compensations for the motor disturbances also suggest that the latter involve less complex, more nearly reflex, integrations than habitual acts. The primary compensations are not by the direct restitution of motor control (recovery from the paresis) but by the acquisition of new patterns of co-ordination which tend to counteract the motor difficulty, much as a normal animal would adapt to a heavy weight attached to one leg. After the destruction of the stimulable cortex the complex adaptive mechanisms are still intact.

These facts argue strongly for a relative independence of the cerebral mechanisms for control of postural activity from those for voluntary movements. The hypothesis which seems best to fit the facts for the rat is that the stimulable area and caudate nucleus are concerned chiefly with the regulation of postural reflexes and that upon these are superimposed the more complex integrations constituting voluntary acts, transmitted to the final common paths by extrapyramidal fibres. This would make the cerebral motor structures an additional stage in the hierarchy of spinal, vestibular, and cerebellar mechanisms for the control of postural reflexes. Such a function has already been ascribed to the corpus striatum by Wilson [18], on the basis of work with monkeys, and the present experiments add to his conclusions only the fact that the stimulable area of the rat is to be classed in function with the striate nucleus rather than with the cerebral habit-mechanisms.

The Function of the Stimulable Cortex in Higher Mammals.

Is such a view of the function of the electro-stimulable cortex contradicted by evidence from the study of higher animals? It is certainly

contrary to the accepted view of its function. In current text-books of physiology and psychology it is frequently stated that the motor area is the (sole) efferent path of voluntary movements from the cortex. Even investigators like Rothmann, who have studied recovery from hemiplegia in animals, look upon the stimulable area as the chief source of the efferent fibres concerned with habitual activity. In speaking of conduction through extrapyramidal paths Rothmann says, "Diese extrapyramidale Leitung dürfte für die Erlernung neuer Bewegungen von grösster Bedeutung sein, während die in festen Besitz des Individuums, übergegangenen gut eingelernten Bewegungen vorwiegend die direkte Verbindung von Grosshirnrinde und Rückenmark, also die corticospinale Bahn, benutzen werden" [16]. Von Bechterew advances a similar view in describing the course of conditioned-reflex arcs across the cortex from the sensory projection areas to the motor area [1].

If this conception is correct for higher forms, it means that a pronounced change in the function of the stimulable cortex has occurred somewhere above the rodents in the evolutionary scale. But such changes in function are rare and in this case the evidence does not seem to show more than that the stimulable cortex of higher forms has taken over a part of the function which the caudate nucleus exercises in rodents.

King [8], using the Marchi method, concluded that the pyramidal tract in the rat is small and unimportant, but Ranson [15] has shown that it is quite large, though made up chiefly of thinly medullated fibres. The area of the tract shown on Ranson's figures is proportionally greater than in primates, so that it cannot be argued that there is any great increase in the anatomical importance of the tract with ascent in the evolutionary scale.

The physiological evidence does not seem sufficient to prove that the pyamidal tracts, even of primates, are the efferent path for impulses to voluntary movement. Fritsch and Hitzig [5] pointed out that the paralysis following destruction of the stimulable area is only partial. Writing of the motor centre they stated that " . . . es ist sicher, dass eine Verletzung dieses Centrum die willkürliche Bewegung des von ihm sicher in einem gewissen Abhängigkeit stehenden Gliedes nur alterirt, nicht aufhebt, dass, also irgend einem motorischen Impulse noch andere Stätten und Bahnen offen stehen um geboren zu werden und um zu den Muskeln jenes Beines zu eilen . . ." Nothnagel observed recovery from the effects of unilateral injury to the motor area and Carville and Duret [4] first showed that the recovery is not due to

the vicarious functioning of the motor cortex of the opposite side. Rothmann [16] showed that in monkeys the extra-pyramidal paths are able to mediate learned activities. Thus it is certain that whatever the normal function of the stimulable cortex in higher mammals it is not absolutely necessary for the performance of voluntary acts.

The chief evidence for the voluntary function of the motor areas comes from the cerebral paralyses in monkeys and man. In them finer manipulative movements are greatly affected and the paralysis is much more severe than in lower mammals, yet even so it is rarely complete. The condition has been characterized as an enormous difficulty rather than an inability to move the paralysed limbs, and there is some evidence that the degree of paralysis varies with the general tonic condition of the organism.

Thus after cauterization of the stimulable cortex on one side the rhesus monkey may show a well-marked hemiplegia. But if he is badly frightened and chased about the room, he uses the limbs of both sides in an apparently normal manner. Left undisturbed, he again lapses into the paralytic condition. Gierlich [6] summarizes other examples of this sort. Similar incidents are occasionally reported for man. A girl with hemiplegia of fourteen years' standing, following diphtheria, with paralysis of the right limbs and marked contractures, tells me that when her brother pushed her from the wharf into a lake recently she used her paralysed limbs actively in climbing out. This patient is mentally retarded and perhaps not reliable. I know of no well-authenticated case of the sort, but evidence of less pronounced effects of emotional disturbance upon paralyses is frequently encountered. Depressing stimuli, such as discussion of the above patient's very distressing home conditions, increase the extent of contracture and limit the power of voluntary movement. Exciting stimuli, on the other hand, will sometimes increase the extent of voluntary movement, and have been used to advantage in re-education (personal communication from Dr. S. I. Franz).

All these facts point to the conclusion that, even in primates, it is not so much the mechanism of voluntary movement which is affected in cerebral paralysis, as some facilitating mechanism whose action is usually essential to the movement, yet is not a part of the direct conditioned-reflex arc. Graham Brown's study of facilitation after section of the connections between pre- and post-rolandic areas [3] shows that the descending fibres from both areas act upon the same final common paths, and that transcortical connections are not necessary for the mutual

facilitation of the areas. Voluntary movements are possible in the absence of the motor areas; there is not conclusive evidence that they function directly in habit; the phenomena of cerebral paralysis can be explained on the assumption that the motor area is postural and facilitating in function; there is clear evidence that the analogous structures in lower forms are not directly concerned with the performance of learned activities. Although not conclusive, these considerations seem sufficient to cast doubt upon the accepted explanation of the function of the motor cortex, and to demand further experimental investigation before we can conclude that the pyramidal tracts form the efferent path for impulses to voluntary movements.

Summary.

It has been shown that the albino rat is able to acquire somæsthetic-motor habits after destruction of the electro-stimulable cortex and the caudate nucleus. Visuo-motor and simple somæsthetic-motor habits which are acquired before the operation are retained after the destruction of these structures and probably after the section of occipito-lenticular fibres. It is clear, therefore, that neither the cerebral-motor areas nor the subcortical nuclei are directly concerned with the performance of learned activities.

Combined destruction of the motor area and the caudate nucleus results in relatively permanent motor disturbances resembling hemiplegia in monkeys. Evidence is given that the difficulty is primarily in assuming new attitudes, and it is suggested that the primary function of the cerebral motor structures in the rat is the regulation of postural reflexes. Existing evidence does not seem to exclude such an explanation of the function of the stimulable cortex, even in man.

REFERENCES.

[1] Bechterew, W. v. "Die Funktionen der Nervencentra," Jena, 1908.

[2] Brodmann, K. "Vergleichende Lokalisationslehre der Grosshirnrinde," Leipzig, 1909.

[3] Brown, T. G. "The Motor Activation of Parts of the Cerebral Cortex other than those included in the so-called 'Motor' Areas in Monkeys, with a Note on the Theory of Cortical Localization of Function," *Quart. Journ. Exp. Physiol.*, 1916, **10**, 103-144.

[4] Carville, C., et Duret, H. "Sur les fonctions des hémisphères cérébraux," *Archives de Physiol.*, 1875, **7**, 352-490.

[5] Fritsch, G., and Hitzig, E. "Ueber die elektrische Erregbarkeit des Grosshirns, *Arch. f. Anat. u. Physiol.*, Jahrg. 1870, 300-332.

[6] Gierlich, N. "Ueber Symptomatologie, Wesen und Therapie der hemiplegischen Lähmung," Wiesbaden, 1913.

[7] Goltz, F. "Ueber die Verrichtungen des Grosshirns," *Arch. f. d. ges. Physiol.*, 1881, **26**, 1-49.

[8] KING, J. L. "The Cortico-spinal Tract of the Rat," *Anat. Record*, 1910, **4**, 245-252.

[9] LANGELAAN, J. W. "On Muscle Tonus," *Brain*, 1915, **38**, 235-380.

[10] LASHLEY, K. S. "Studies of Cerebral Function in Learning," *Psychobiology*, 1920, **2**, 55-135.

[11] LASHLEY, K. S. Studies, &c., II: "The Effects of Long-continued Practice upon Cerebral Localization," *Journ. Compt. Psychol.*, 1921.

[12] LASHLEY, K. S., and FRANZ, S. I. "The Effects of Cerebral Destruction upon Habit Formation and Retention in the Albino Rat," *Psychobiology*, 1917, **1**, 71-140.

[13] LUCIANI, L. "Human Physiology," London, 1915, vol. 3.

[14] MONAKOW, C. v. "Die Lokalisation im Grosshirn," Wiesbaden, 1914.

[15] RANSON, S. W. "The Fasciculus Cerebro-spinalis in the Albino Rat," *Amer. Journ. of Anat.*, 1912-13, **14**. 411-424.

[16] ROTHMANN, M. "Ueber die physiologische Wertung der cortico-spinalen (Pyramiden-) Bahn," *Arch. f. Anat. u. Physiol.* (Physiol. Abth.), 1907, 217-275.

[17] SHERRINGTON, C. S. "Postural Activity of Muscle and Nerve," *Brain*, 1915, **38**, 191-234.

[18] WILSON, S. A. K. "An Experimental Research into the Anatomy and Physiology of the Corpus Striatum," *Brain*, 1913-14, **36**, 427-492.

[19] WILSON, S. A. K. "On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits," *Brain*, 1920, **43**, 220-268.

A PSYCHOLOGICAL INQUIRY INTO THE NATURE OF THE CONDITION KNOWN AS CONGENITAL WORD-BLINDNESS.

BY LUCY G. FILDES.

THE aim of the investigation about to be described was to discover something of the psychological characteristics of the condition commonly called by the misleading term of congenital word-blindness—a condition which shows itself most clearly in the subjects' extreme difficulty, or even total failure, in learning to read, and appears to be closely related to the various forms of acquired alexia met with commonly as the result of brain injury in later life.

Three explanatory theories, it will be remembered, have been put forward as indicating the nature and cause of this condition, viz.:—

(1) A theory which assumes the existence of definitely localized and circumscribed visual and auditory word-centres in the brain, the destruction or isolation of which will destroy language in either its visual or its auditory aspect;

(2) A theory which interprets word-blindness as only one symptom in a general lowering of mental ability; and

(3) One which attributes the condition to a more specialized lowering of power in the primary visual centres, rendering true visual perception of words and of other complex sense-data difficult.

The main problems raised by these different interpretations are two in number. Is inability to learn to read or the loss of the power of reading due to specific or to general defect? If the former, does the defect show itself only in reading, or does there appear to be any general lowering of visual power? A psychological investigation should throw light on these points.

The subjects mainly used for the investigation were twenty-six in number—all children between the ages of 9 and 16 years, who were in attendance either at ordinary elementary schools (4) or at special schools for mentally defective children (22). They were selected on the report of their teachers as finding reading a very great difficulty. Before the work peculiar to the investigation was begun, all the children were tested (*a*) with the Stanford revision of the Binet scale, in order to get

some idea of their general mental capacity, and (*b*) with various recognized reading tests, in order to get an estimate of their reading power.

The results of these tests show that the subjects differed very considerably in general ability as measured by the Terman scale—the intelligence quotients varying from 50 to 111. Classification on the basis of these results gives the following groups :—

1 subject has superior intelligence	I.Q. = 111
4 subjects belong to the class dull and backward	I.Q. = 82-88
8 subjects show border-line deficiency	I.Q. = 70-79
13 subjects are classifiable as morons of different grades ..	I.Q. = 50-69

Four of the subjects from the first two groups were in normal schools, the fifth in a school for the mentally defective. It is interesting to note that this boy fell below the others of the same group, chiefly in reading ability. He was, however, better at reading than the brightest boy, who showed superior intelligence.

Testing for reading power in particular revealed also a great variation in the ability of the subjects. Some of them could recognize no single letter, word or figure with any certainty; others could read simple words and knew all letters and figures well. It was estimated, however, that no child was less than four years retarded in reading ability and some of them showed retardation much greater than this. Even the children who could read best had a marked difficulty in writing from memory words which they could read—they could not spell.

No relationship existed between the subjects' intelligence quotients and their power in reading. Two of the worst readers were the least intelligent and most intelligent boys. The three worst cases examined, i.e., cases with no reading power at all, had intelligience quotients of 61, 79 and 78 respectively. Many defective boys with such high intelligence quotients read quite well.

The tests in the scale used for finding the intelligence quotients gave no indication of any special disability common among the children. On the whole they failed most on tests requiring auditory repetition, but this characteristic does not mark them off from defective children who can read.

The main suggestion of the preliminary work, then, is that inability to learn to read depends rather on a specific than on a general defect, although in school life such a defect may, because of its nature, simulate one more wide-spreading in character.

Experimental Work.

The experiments aimed at investigating each subject's ability in the various forms of mental activity known to be involved in the complex act of reading, in so far as that act implies the power to recognize words, letters and figures previously taught. Reading proper, i.e., reading new words, involves, of course, yet further powers, in particular the power of ready analysis and synthesis and quickness in applying what is known to new situations; and for this reason probably most defective children are slow in reading and in learning to read. It was, however, in their failure to retain what had actually been taught that our group of non-readers showed their extreme peculiarity, and because of this fact the experiments aimed primarily at testing activities required here. These activities fall into three groups:—

(1) Rapid and easy visual discrimination of forms, together with the ability to retain these without undue repetition.

In particular this discrimination must be extended to forms which closely resemble each other (e.g., ran, ram), and also to those which differ only in the arrangement of their parts, (e.g., b, d, p—god, dog—saw, was—&c.). It may here be noticed in passing that this power of retaining the knowledge of the relationship of the parts to each other and to the whole in a visual percept is one often late in development.

(2) A similar rapid and easy discrimination of sounds.

(3) The possibility of establishing readily an association between a given form and a given sound.

Hence the experiments used can also be arranged in three groups according to the special power which they aimed at testing. Each was carried out with most of the members of the non-reading group, although it was not always possible to include them all. In each experiment the results of the group of non-readers tested are compared with those of a group of readers, equated as far as possible with the non-readers with regard to age and mental ability.

All the experimental work was carried out individually with each child, the procedure being the same throughout the experiment in all cases.

§1.—*Experiments to test the Powers of Visual Discrimination and Retention.*

Experiment 1.—The aim of this experiment was to test the subject's ability to distinguish between unlike and to recognize like forms when these are presented in sequence. In particular it was planned to reveal whether and how far—

(1) Forms showing right and left or top and bottom reversal can be distinguished in memory, and

(2) Forms containing common elements tend to be confused.

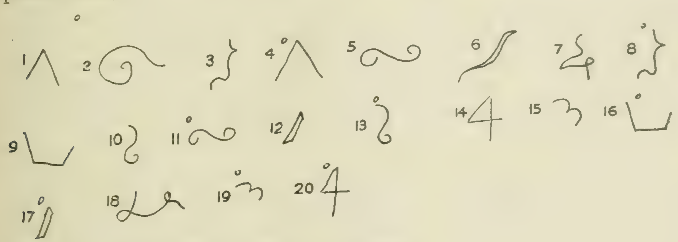
Eighteen series of forms were prepared, each series containing some quite different and some similar or like forms. The number of forms in each series was twenty; of these eight were either exactly like or closely resembled others. The same form was never used in more than one series. Each series was taken as a separate unit and the subjects were told that they were to make their judgments in relation to the forms of that series only.

The forms were shown to the subjects individually, each being exposed for a period of two seconds. After each exposure the subject was required to say "Yes" if he judged the form to be like one already seen in the series, and "No" if he judged it to be a different form.

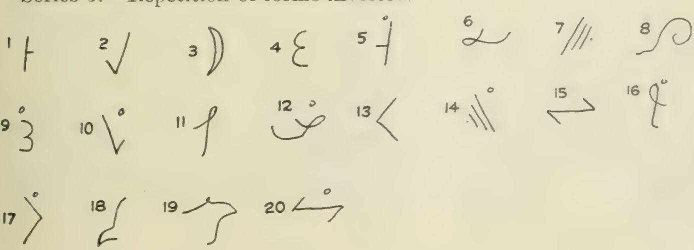
Care was taken to prevent the repeated forms from occupying similar positions in the different series. As a rule, two series of forms only were exposed during one sitting with each subject. In this way, the possibility of confusion with forms of other series was reduced; as a matter of fact there is no evidence to show that such confusion constituted a source of error.

The following are examples of the forms used:—

Series 1.—Repetition of like forms. (° Indicates that the form is a repeated one.)



Series 3.—Repetition of forms inverted.



The repeated forms in the different series were :—

Series 1 and 2. Exactly alike.

Series 3 and 4. Alike except for RL inversion.

Series 5 and 6. Alike except for TB inversion.

(After passing judgment on the foregoing series, the subjects were told that the differences in direction were to count as points of dissimilarity.)

Series 7 and 8. Alike except for TB inversion.

Series 9 and 10. Alike except for RL inversion.

Series 11, 12 and 13. Alike except for either TB or RL inversion.

Series 14, 15 and 16. Alike either wholly or partially.

Series 17 and 18. Alike either wholly or in one part—the whole form consisting of three parts.

The results, given in percentages, are tabulated below. Fourteen non-reading boys worked through the whole eighteen series. Twenty-four non-reading boys and seven reading boys (i.e., boys who could read up to or beyond their intelligence level), also defective, worked with the first two series also.

TABLE I.—SHOWING THE ABILITY OF NON-READING BOYS TO RECOGNIZE LIKE FORMS AS LIKE, AND DIFFERENT FORMS AS DIFFERENT.

No. of series		Percentage of answers "Yes" to forms entirely alike or alike in some particular		Percentage of answers "Yes" to forms entirely different
1	..	82·5	..	5·5
2	..	65·8	..	2·7
3	..	66·6	..	1·6
4	..	56·6	..	5·0
5	..	60·7	..	5·3
6	..	59·8	..	5·3
7	..	47·3	..	6·5
8	..	48·2	..	2·4
9	..	50·0	..	2·9
10	..	45·0	..	6·5
11	..	42·8	..	4.8
12	..	45·5	..	6·5
13	..	55·7	..	7.0
14	..	+ 69·2 and °13·4	..	6·4
15	..	+ 51·9 and °28·0	..	7·7
16	..	+ 32·7 and °26·9	..	4·4
17	..	+ 38·0 and °15·0	..	13·9
18	..	+ 51·9 and °25·0	..	5·1

+ = Whole form repeated. ° = Part repeated.

TABLE II.—SHOWING COMPARATIVE ABILITY OF READERS AND NON-READERS IN RECOGNIZING LIKENESS IN FORMS SUCCESSIVELY PRESENTED.

	Percentage of answers "Yes" (1) To like forms	Percentage of answers "Yes" (2) To unlike forms
Series 1 :—		
Readers	57·0	3·5
Non readers	76·0	6·9
Series 2 :—		
Readers	43·0	6·0
Non-readers	61·0	2·4

These results seem to warrant the following inferences :—

(1) All the children examined were able with considerable ease to recognize like and to distinguish unlike forms successively presented, when the forms were like or unlike in all particulars. The non-readers were in no way inferior here to the readers.

(2) The non-readers found forms alike except for differences of orientation very difficult to distinguish, even when special attention was directed to that aspect of the form.

(3) Difficulty was also occasioned by repetition of a part of a form in other surroundings.

It was unfortunate that the other series could not be worked through with the reading group. Reference to a similar experiment, however, performed with a group of younger subjects, suggests that the difficulty shown here by the non-readers in dealing with forms differently orientated is considerably in excess of the normal. The results of this younger group show only 26·4 per cent. and 23·7 per cent. of error in dealing with two like series.

Experiment 2.—This experiment was planned to discover whether the length of exposure of a form made any difference to the children's power to recognize it on a second presentation, and if this did occur to see whether the non-readers in particular suffered from shortening of the exposure.

The subjects were fifteen non-readers and ten readers.

The work was similar to that of Experiment 1, a series resembling in construction Series 1 and 2 of that experiment being used. It differed only in that here, by the use of a simple drop-shutter apparatus, the exposure-time of each form was momentary, instead of being, as in the first experiment, two seconds in length.

TABLE III.—SHOWING COMPARATIVE ABILITY OF READERS AND NON-READERS IN RECOGNIZING LIKE FORMS UNDER CONDITIONS OF MOMENTARY EXPOSURE.

	Percentage of (1) Correct answers	Percentage of (2) Incorrect answers
Readers ..	45·0 ..	19·0
Non-readers ..	56·0 ..	10·0

There is no evidence here that non-readers as a class fail in speed of perception or in ability to recognize forms with material of this type.

Note.—In spite of previous practice with the apparatus all the children found this work much more difficult than the related work in Experiment 1, and it is not felt that the results are very trustworthy, since certain individuals, finding the work beyond their powers, tended to respond automatically—saying "Yes" or "No" to all the forms. Cases in which this tendency operated throughout have been omitted from the results.

Experiment 3. *Reproduction of forms.*—In order to test further the effect of length of exposure on the ability to perceive a form in the two selected groups of subjects, an experiment was planned which demanded the reproduction of forms seen only with momentary exposure.

The forms were exhibited by means of the apparatus already mentioned, each form being shown three times in succession. After each exposure the subject was required to draw what he had seen. Finally the subjects copied each of the drawings from the original card.

Twenty boys, ten readers and ten non-readers, performed this experiment.

It is in practice impossible to judge the results by any allocation of marks, but the general impression gained by a careful examination of the drawings is that there is nothing to distinguish the work of the non-reading group of boys from that of the readers. Some of both groups reproduce well and some reproduce badly. A much higher correlation exists between the subjects' reproductions and their drawings from the copy than between their reproductions and their ability to read.

We seem, then, justified in inferring that slowness in visual perception is not an important factor in causing difficulty in learning to read.

Experiment 4. *An experiment in learning from visual material.*—The aim of this experiment was to discover whether the boys grouped as non-readers showed any special disability in learning from visual material, the method of learning being left free.

To this end four large squares were prepared, each divided into nine small squares. Each small square was filled with either (1) a colour (all the subjects could name every colour used quite accurately) ; or (2) a simple geometrical drawing or a representation of some common object.

Each large square was shown to the subject for a fixed period (thirty seconds). He was then required to indicate on a duplicate skeleton square the contents of each small square.

This experiment was performed by thirty-six boys, twenty-four non-readers and twelve readers.

The filled-in duplicate squares were marked as follows :—

(1) Each form or colour correctly placed received one mark ; (2) each form or colour correctly given but misplaced by not more than one square in any direction received half a mark.

The marks gained by the respective groups are given below :—

TABLE IV.—RESULTS OF THE COLOUR SQUARE EXPERIMENT.

The figures given indicate the percentage of actual in relation to possible successes with each square, each group of boys being taken as a whole.

Number of square :	1	2	3	4
Readers ..	45 ..	38 ..	43 ..	52
Non-readers ..	44 ..	39 ..	49 ..	42

Examination of these results makes it quite clear that from this experiment no evidence can be obtained which would indicate that non-readers find learning from visual any more difficult than do readers of equal mental attainments. The percentage of successes in the two groups are almost identical.

In all cases, however, the method of learning employed was audito-motor-visual.

Further, success in this experiment did not depend on the power to differentiate between forms nearly alike or forms differing only in position. Results were considered as right if the drawings made were recognizable, irrespective of the accuracy of their details. Nearly 50 per cent. of the errors made were due to misplacement of the colours or forms, while many of the drawings showed faulty orientation.

Experiment 5.—In common with the two succeeding experiments, Experiment 5 aimed at carrying further the investigation already begun in Experiment 4, i.e., it was planned to discover whether the reading and non-reading groups differed in their ability to learn from visual material. These three experiments, however, differed from Experiment 4 in that—

(1) The material used (digits and words) was actually of the kind ordinarily used in reading. Learning from it, therefore, demanded the power of discriminating rapidly between very similar visual presentations.

(2) The actual learning was more definitely visual in type. As far as possible auditory and motor aids to learning were prevented.

The fact that material such as digits and words could be used for experimental purposes with children defined as non-readers requires perhaps some explanation. The tests of reading ability were made at the beginning of the experimental work; these tests were made near its end, some nine months later. In the meantime attempts to teach reading had been carried out side by side with the actual experiments. No child had been found entirely incapable of learning any words or figures when taught alone in this way. At the end of the nine months, therefore, all the subjects used for these three experiments could name all the words and figures employed in them quite well.

In Experiment 5 digits were used as learning material. A typed list of numbers was prepared, having on it four 3-place, four 4-place, and four 5-place numbers. These were exposed in turn to the subject, each for a period amounting to one second for each digit. Immediately after each exposure the subject was required to give the number seen.

The work was done by eleven readers and twenty-six non-readers.

The results were marked in terms of error as follows:—

(*a*) Omission or insertion of a digit, or the substitution of one digit for another = 1 error.

(*b*) Misplacement of a digit by not more than one place = ½ error.

(*c*) Interchange of two neighbouring digits = ½ error.

TABLE V.—SHOWING AVERAGE ERROR PER BOY IN THE REPRODUCTION OF DIGITS LEARNT VISUALLY.

Number of digits in group :	3	4	5	Total
Readers	0·23	1·27	3·3	4·8
Non-readers	1·9	4·6	8·8	15·3

A glance at the table given above is sufficient to indicate the comparative failure of the non-reading group here. An examination of the nature of their errors is even more instructive. The chief causes of error were :—

(*a*) Misplacing of digits in the number given, and

(*b*) Misreading of digits similar in form, e.g., 6 and 9, 5 and 8, 5 and 3, 3 and 8, &c.

The results of this experiment, therefore, seem to confirm those

found in Experiment 1, viz., that non-readers as a class find difficulty in distinguishing between visual impressions which closely resemble each other, although they are readily able to appreciate and to retain more marked differences.

Experiment 6.—To test the power of reproducing numbers presented visually with momentary exposure.

The main aim of this experiment has already been indicated (Experiment 5). Its further object was to test the effect of length of exposure on the reproduction of numbers.

The numbers used were printed on cards and exposed in the drop-shutter apparatus previously mentioned. Five exposures of each number were made, the subject's reply after each presentation being noted. The numbers used were :—

(1) 71	(4) 65	(7) 27408	(10) 914	(13) 3502
(2) 92710	(5) 7930	(8) 049	(11) 36019	
(3) 629	(6) 5186	(9) 4917	(12) 831	

TABLE VI.—SHOWING THE RELATIVE ABILITY OF READING AND NON-READING BOYS IN THE REPRODUCTION OF NUMBERS EXPOSED MOMENTARILY.

Group A.—Twelve Readers.

No. of exposure :	1	2	3	4	5
Card 1	2	0	0	0	0
2	25½	17½	10	7	2½
3	15½	5	2½	2½	3
4	4½	2½	3½	1½	1
5	14½	10½	11	7	5
6	9½	5½	4	2½	3
7	12	8½	2	1	0
8	8½	2	1	1	2
9	27½	14½	11	7	6½
10	2½	2	1	1	1
11	12	7	5	5½	2
12	3	2½	2	2	2
13	15	7½	4	3½	3½
Total	152	85	57	41½	31½
Average error per head	12·6	7·0	4·7	3·4	2·6

Group B.—Ten Non-readers.

No. of exposure :	1	2	3	4	5
Card 1	6½	2	1½	1	0
2	29½	20½	15	13½	7½
3	16	15	11½	15½	10½
4	6½	5	5	2½	2
5	30	23½	13½	13	8½
6	9½	9½	6	4½	5½
7	21½	10	8	5½	4
8	3½	1	0	0	0
9	25½	16½	12½	8½	10
10	9	5	4	3	3
11	4	1	1	0	0
12	14	12	9½	10½	6½
13	13	12½	10½	10	7
Total	187½	133½	98	87½	64½
Average error per head	18·7	13·3	9·8	8·7	6·4

The results were marked as in the preceding experiment. The number of errors on each successive exposure (taking all the digits together) was then found for each group of boys. Finally, the average error per head per exposure was estimated for the two groups.

Table VI gives the actual results.

These results entirely support the inferences made from the preceding experiment, for the inferiority of the non-reading group is evident. It is hard to say, however, how far the comparative failure of this group is due to the difficulty previously noted, their inability to perceive small differences in visual presentations. That this is a difficulty is clear, but that it is the only or even the main difficulty is doubtful. The children's behaviour in this experiment in particular suggested that part of their failure was due, not only to their inability to retain the visual impression, but to the slowness of their association processes, which prevented their getting sufficiently quickly the name of the number presented.

Experiment 7.—To test the subject's ability to recognize words and letters shown under conditions of momentary exposure.

The material used in this experiment consisted of the following words and nonsense words: (1) window; (2) char; (3) bok; (4) floor; (5) feg; (6) tadel; (7) flore; (8) cair; (9) pen; (10) bo; (11) wall; (12) box; (13) table.

They were printed separately on cards and exhibited in the drop-shutter apparatus. Three exposures were made of each word, the subject being required to state what he had seen after each exposure.

Nine readers and ten non-readers performed the experiment. The ten non-readers could, however, recognize under ordinary conditions all the letters and all the words used.

The results were marked on a scheme similar to that used in the preceding experiment. Table VII gives the percentage of error in the two groups:—

TABLE VII.

	No. of exposure:	1	2	3
Words and non-words	Readers	35	21	22
	Non-readers	38	23	13
Words alone	Readers	26	16	14
	Non-readers	27	19	10
Non-words alone	Readers	59	34	30
	Non-readers	53	27	18

Contrary to the results of the preceding experiments, no indication is given here of any difference in ability between the reading and

non-reading groups. Two reasons may be offered to explain this fact:—

(1) Since the non-reading children had learnt these particular words and letters, they may, for purposes of this experiment, be considered readers. This argument could, however, be equally applied to the work with digits.

(2) It is well known that very little is actually seen under conditions of momentary exposure—what we do see is added to through association and depends very largely on what we expect. This being so, the group of non-readers was at a considerable advantage in this experiment, for they knew only a few words and expected those words to occur in any reading exercise. The readers, on the other hand, had a much wider range of words called up by what they saw, and so were the more liable to error.

It is now possible to summarize certain general inferences that can be made with regard to the power of non-reading children in visual discrimination and learning. Comparing them with readers of equal mental ability, we find that these children are able:—

(1) To discriminate between unlike forms and to remember them in a normal degree, except in cases in which the forms (*a*) differ only in part or (*b*) are alike, except in their orientation.

(2) To make such discriminations equally with the normal, whatever the length of exposure time of the form.

(3) To learn from visual material also in a normal degree when the method of learning is free and the forms easy to distinguish, but

(4) To find such learning abnormally difficult in cases in which it is (*a*) more exactly visual and (*b*) concerned with easily confused material, e.g., digits.

The main reasons suggested as a cause for this trouble are slow discrimination of similar visual presentations and faulty association between a visual impression and its appropriate name. Probably both causes operate in every case.

§ 2.—*Experiments to test Auditory Discrimination and Retention.*

Experiment 8. *The auditory learning of digits.*—The work of certain of the non-reading subjects had indicated that possibly a specific auditory difficulty existed in some cases of inability to read. The two following experiments were, therefore, planned in order to test the power of the non-reading group as a whole in auditory discrimination and retention. In this first experiment the material used was digits—

in number and arrangement exactly similar to those used in Experiment 5, i.e., four 3-place, four 4-place and four 5-place numbers were employed.

The numbers were read by the experimenter at a uniform rate of one digit per second and immediately after the completion of each a reproduction was attempted by the subject.

The results were marked according to the scheme given in Experiment 5 (p. 293). They are recorded below.

TABLE VIII.—SHOWING AVERAGE ERROR PER BOY IN THE REPRODUCTION OF DIGITS LEARNT BY HEARING.

Number of digits in group:	3	4		Total
Readers	0	0·13	2·6	2·73
Non-readers	0·12	0·94	4·44	5·5

The non-readers as a group have greater difficulty in reproducing digits presented auditorily than do the readers, although their difficulty is not relatively so great as with visual presentation of similar material.

The work with digits was carried out by exactly the same boys in both groups—readers and non-readers.

Experiment 9. *The auditory learning of sentences.*—The aim of this experiment was to test the power of learning among the non-readers from auditory material other than digits.

Twenty-four sentences were used, including four with each of the following number of syllables: 10, 11, 12, 13, 14 and 15 syllables. Each sentence was read aloud to the subject and an immediate reproduction required.

The results were then marked according to the following scheme:—

(1) Insertion or omission of any word = 1 error.

(2) Misplacement of a word or phrase = ½ error.

The subjects were twenty-three non-readers. The value of the results was estimated by comparison with what is normal for the mental ages of the children concerned.

The actual numerical results (in terms of error) are given in Table IX (page 299).

Most children of from 6 to 7 years of age are able to reproduce accurately sentences of from sixteen to eighteen syllables. Almost all these subjects had a mental age of over 7 years—a fact which indicates that their difficulties in this experiment were far greater than would be expected.

These results, therefore, are in agreement with those of the preceding test—the non-readers as a group show a special disability in retaining and reproducing a series of auditory impressions.

TABLE IX.—SHOWING THE NUMBER OF ERRORS MADE BY EACH SUBJECT IN THE REPRODUCTION OF SENTENCES.

Number of syllables in each sentence		10	11	12	13	14	15
Total number of syllables to be reproduced in group of four sentences		40	44	48	52	56	60
Errors made by subject	(1)	3	2	15	7	7½	22
	(2)	0	0	0	2	2	5
	(3)	2½	3	5	6	11	26
	(4)	0	1	8	3	10	19
	(5)	0	2	2	2	7	15
	(6)	1½	1	10½	5	15½	34
	(7)	2	1	3	6	11	16
	(8)	2	5	8	3	18	25
	(9)	0	0	0	1	6	11
	(10)	0	0	0	4	0	3
	(11)	0	0	0	0	1	0
	(12)	0	0	0	0	0	0
	(13)	0	1	2	1	1½	2
	(14)	0	0	1	2	5	7
	(15)	0	0	4	2	0	5
	(16)	0	0	0	0	0	2
	(17)	2	0	3	0	1	9
	(18)	5½	3	14	14	19	32
	(19)	0	0	0	1	1	2
	(20)	1	0	0	0	6½	6
	(21)	0	0	2½	3	6	10
	(22)	0	0	0	0	1	2
	(23)	1½	9	19½	11	18½	39

Certain points are, however, of special interest:—

(1) Individual differences in capacity revealed by this experiment are very great, much greater than those shown when visual material was used. These differences show no correlation with variations in general mental ability.

(2) Certain subjects whose auditory power as measured by these experiments is very limited, are among the worst readers in spite of the fact that their general mental capacity is comparatively high.

(3) No correlation exists between the findings in the auditory tests and those of certain visual tests, e.g., Experiment 1: correlation between results of Experiments 1 and 8 = ·073, between Experiments 1 and 9 = −·21.

With regard, then, to the auditory capacity of the non-reading group, we may say:—

(1) That the non-readers as a group show a lowering of auditory power, but that their difficulties in dealing with auditory material are not, on the whole, so great as their difficulties with visual material.

(2) No correlation exists between auditory power as measured by the tests and certain forms of visual power. Defect in either capacity

seems to be specific rather than an expression of a general lowering of ability.

(3) That certain individuals show auditory difficulties to a peculiarly marked degree. These individuals are especially bad in reading.

§3.—*Experiments to test the Power of making Audito-Visual Associations.*

We have now to consider the experiments in the final group.

Experiment 10.—The aim of this experiment was to try to estimate the subject's ability to form an association between :—

(*a*) A given form exhibited visually ; and

(*b*) A common name.

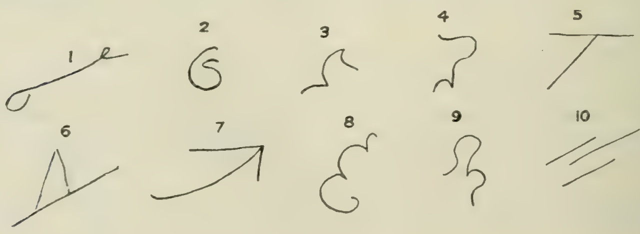
Three sets of forms were prepared—ten in each set—somewhat similar to the forms used in Experiment 1, except that in this case there were no repeated forms. Corresponding to each set of forms a list of ten common names was made.

The subjects were shown the forms each in turn for a period of two seconds—the experimenter in the meantime saying, and the subject repeating, the associated name. Immediately after the last form had been shown, the forms were shown again in an order different from that originally used, and the subjects were required to give their associated names. If the subject failed to give the name after a period of two seconds, it was again given by the experimenter and repeated by the subject. The forms correctly named were put on one side and not given again. This procedure was continued until all the names were known.

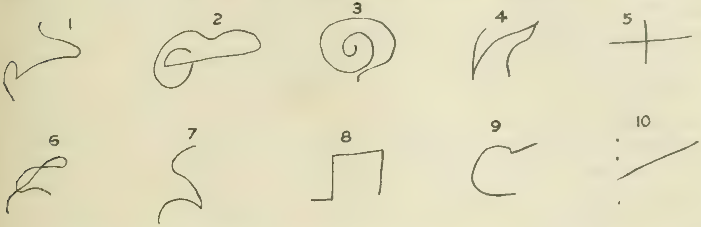
Illustrations of the forms are given below.

Series 1. *Names :* (1) dog ; (2) potato ; (3) waggon ; (4) frog ; (5) train ; (6) grass ; (7) chair ; (8) coat ; (9) moon ; (10) window.

Forms :



Series 2. *Names:* (1) shoe; (2) mud; (3) sun; (4) boat; (5) road; (6) tree; (7) post; (8) button; (9) sheep; (10) pond.



The experiment was carried out by twenty-six readers and thirteen non-readers, and the result is shown in Table X.

TABLE X.—SHOWING THE PERCENTAGE OF FORMS CORRECTLY NAMED AT EACH SUCCESSIVE EXPOSURE, THE RESULTS OF (*a*) ALL THE READERS AND (*b*) ALL THE NON-READERS BEING TAKEN TOGETHER.

	Series 1						Series 2					
Exposure	1	2	3	4	5	6	1	2	3	4	5	6
Readers	59	18	11	6·9	3	—	46	20	17	6	3	—
Non-readers	50	20	18	7	2	—	48	23	10	10	2	2

Clearly there is from this experiment no evidence to indicate that the non-readers find association of this type any more difficult than do the readers. In fact, two of the worst readers (boys unable to read any words and in one case any letters or figures) make two of the best scores, whilst the worst score but one is made by a boy who reads fluently.

Certain subsidiary points are important, viz.: (1) There is no evidence to show that the place occupied by the form in the original series has any influence on the ease of recall of the associated name; rather (2) the relative ease of reproduction of the associated name seems determined to a large degree by the nature of the form. This is well shown in Table XI.

TABLE XI.—SHOWING THE RELATION BETWEEN—

(*a*) The nature of the form, and
(*b*) The number of successes gained on the first attempt at naming.
(All the subjects taken together.)

Series 1—										
Number of form ..	1	2	3	4	5	6	7	8	9	10
Number of successes on first naming ..	17	27	9	16	27	23	28	22	10	28
Series 2—										
Number of successes on first naming ..	12	14	18	9	22	7	6	20	12	16

The fact that the possibility of remembering the associated name depended, at least in part, on the nature of the form is a particularly interesting one. That it did so depend seems clearly indicated by the results. Forms 7, 10, and 5 of Series 1, and Forms 5 and 8 of Series 2 could not otherwise have shown to so great an advantage. The names were all ordinary names and it is unlikely that one or two of them should have meant so much more than the rest to a number of children with widely differing environments as to account for the results shown. No child was in the least unfamiliar with any name and the names were remembered almost always when the association was forgotten.

It is difficult to draw any definite conclusions as to what form characteristics rendered naming easy; but it certainly appears as if one important factor was the shape of the forms concerned. A form which could be mentally described by the subjects seemed to be most easily named. Those worst named (Series 1 to 3, Series 2 to 6 and 7) were all of an indefinite shape, not readily to be described in words.

And the accounts of how they remembered given by the subjects lend colour to this view. "There were three lines." "It had dots and a line." "It was round," &c.

The most successful associations, then, were not simple direct associations between a given visual impression and a given sound. They were associations between a visual impression which had meaning —which was consciously analysed and its parts related definitely to each other—and a sound also full of meaning. For even more often in the records is there indication that the association depended for its retention on the meaning of the word as well as on that of the form, e.g., Series 1 to 2 is said to be remembered "because a potato is round"; Series 2 to 5 "is like a road"—"it is straight"—"it is a cross —cross-roads," while No. 8 of the same series is known "because it's nothing like a button," "a button isn't that shape," &c.

Suggestive, too, is the behaviour of the subjects. They all enjoyed the experiment and many of them were amused by it. But the attitude of any individual in accepting the associated names for the first time differed very considerably, apparently depending on whether he did or did not consider that the name fitted the form. To some forms the attitude was one of doubt or question, as if the name had not quite been heard; in the case of others there was a ready acceptance of the name and a look of pleasure. "Button" frequently induced laughter.

This difference, i.e., the fact that the nature of a form, either

because of its shape or because it fits itself readily on to the associated sound or the meaning of that sound, determines the ease with which the associated sound is retained—is, of course, nothing more than is generally found with children in learning letters. Therefore, some letters, (e.g., o), are particularly easy while others are hard.

Summarizing the conclusions that can be drawn from these facts it appears :—

(1) That in forming an association between a visual presentation and a name, the element of meaning is of primary importance.

(2) That this meaning factor may be inherent in the form itself, or may depend on the nature of the form considered in relation to the meaning involved in the name.

(3) That the less distinctive the forms are in themselves the less easy also is the retention of their associated names; and that therefore

(4) Any difficulty which leads to failure to grasp fully the details of presented forms or to inability to distinguish between them will be likely to render verbal associations with them difficult also.

Experiment 11. *The association of words and Greek letters.*—This experiment aimed at testing the subject's capacity to form associations between common names and letter forms, i.e., it aimed at testing the hypothesis formulated from the results of the last experiment, that the meaning factor inherent in a form is an essential factor in establishing verbal associations with that form.

The material used resembled that used in Experiment 10, except that Greek letters were substituted for the more distinctive forms. The method of procedure was also the same as before.

The letters used were (1) γ; (2) δ; (3) η; (4) λ; (5) μ; (6) ν; (7) ξ; (8) ψ; and the names (1) cow; (2) river; (3) flower; (4) box; (5) wall; (6) paper; (7) rain; (8) hen.

The results show that here the associations were harder to make than in the preceding experiment. In spite of the fact that only eight letters were used as against ten forms, the percentage of successful responses on the second showing of the letters was considerably less than it had been in Experiment 10, i.e., 41 per cent. as against 51 per cent.

The subjects themselves said that the work was harder because the forms were "like each other." There was a constant tendency to confuse "cow," and "box" and "cow" and "paper," owing to the similarity of the forms γ and λ and γ and ν.

It was more difficult in this experiment than in the former to get

any indication from the subjects themselves as to what memory depended on. Usually they declared that they did not know how they remembered. There is, however, some evidence of the influence of meaning. γ was said to be "like horns," ξ to "look like rain," &c.

We find then that the results of this experiment seem to give support to the conclusions previously drawn. They show clearly that it is more difficult to form associations between common names and forms which closely resemble each other, not having in themselves any definite characteristics, than between similar names and more distinctive forms.

Experiment 12. *Learning the names of eight Greek letters.*—An attempt was now made to estimate the subject's power to establish a connection between eight Greek letters and their proper names. The procedure was identical with that of the preceding experiments.

The letters used were α, β, σ, ρ, ζ, ϕ, π, θ.

There is great difficulty in estimating the value of the results in this case, for various reasons, viz.:—

(*a*) Often the subject was quite unable to say the name after it had been said by the experimenter. Consequently it had to be repeated—sometimes more than once. This fact meant that the time of the original exposure in this experiment was much longer than in the two preceding ones, and a repetition factor, not present with them, was involved.

(*b*) When the answers (i.e., the associated names) were being given by the subjects, it was often impossible to tell whether the right answer was being given or not. This was particularly so with many of the boys, who could not, or did not, make correctly such sounds as "th," &c.

(*c*) A considerable tendency to automatism was shown, i.e., one sound (possibly a letter name) was given in answer to all the letters shown. This tendency was due evidently to the felt difficulty of the task. It provides an answer without effort when too great an effort is demanded.

Bearing in mind these facts, with the accompanying result that the children's efforts were judged too leniently, it is interesting to see that here the number of successful responses to the second exhibition of the letters is reduced from 41 per cent. in Experiment 11 to no more than 17 per cent.

A marked difference, also, was shown in the attitude of the subjects. There was little enjoyment. The boys looked worried and baffled, as

if they were up against a task which they could not accomplish. It was often hard to retain their attention. The tendency to automatism has already been noted. Another tendency that was very prevalent was the changing of the name of the letter to one somewhat similar which had meaning to the subject. For example, ρ became "road," ϕ became "fine," and σ was called "signal." The similarity of many of the names led to great confusion; β, θ, ζ were continually muddled, all of them being called indiscriminately "beta" or "meta."

It is interesting to note that 44 per cent. of the successes on the second exposure of the letters were with π, and 32 per cent. with β, which, as one subject justly remarked, "was like B." No correlation exists between the boys' success in this experiment, and in Experiment 10 ($r = 0·05$), but a fairly high correlation is shown between its results and those of the experiment testing the power of immediate reproduction of digits presented auditorily ($r = 0·63$; $PE = 0·16$).

Since, therefore, the only point in which this experiment differed from the preceding one lay in the nature of the name or sound, it is evident that the ease of association between a visual presentation and a sound depends partly on the nature of the sound. The important points with regard to the sound which determine success in reproduction seem to be:—

(*a*) That it shall have meaning.

(*b*) That it shall be readily distinguishable.

Summarizing the conclusions that can be drawn from the three allied association experiments, we find:—

(1) That the non-readers as a group make associations between meaningful material (i.e., dissimilar and characteristic forms and words with meaning) as readily as do the readers; but that their difficulty is increased at a rate in excess of that of the readers with the increase in similarity between the forms and sounds presented.

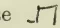
(2) That in all cases the ease with which an association is established between a given form presented visually and a given name depends on the nature of both name and form, and

(3) That the two points of importance with regard to both form and sound in making for ease of association are: (*a*) that they shall be readily distinguished from each other; (*b*) that they shall have meaning, apart from the meaning inherent in their actual presentation.

It is, of course, easy to see that these two points are but one aspect

of the same thing. The form [is easier to distinguish from other forms and also to remember than the form  ; because by its very nature it has more meaning: "It has three lines and two angles"; "It is two horizontal lines joined by a vertical line on the left"; "It is a square with one side left out"; "It is nearly a capital E," &c. In the same way "cow" has more meaning than "beta," although in this case the meaning is not inherent in the form of the word.

Hence cow and [are more easily retained than  and beta.

Further,  and button can be associated because  "is not like a button," and γ can be associated with cow because "a cow has horns"; whereas between  and button, and between  and beta no links of attachment are likely to occur to the mind.

In the same way, when the figure 6 is known, the letter b may be successfully learnt in spite of years of failure if it is realized that the "bee has six legs."

We see, therefore, that any difficulty which hinders a rapid grasp and differentiation of forms and sounds will be likely to hinder their retention and their association, since the hooks of memory will in such a case be either absent or few in number.

Summary.

It is now possible to estimate the bearing of the experimental work which has just been described on the theories commonly held as explanatory of the condition having as its most obvious symptom failure to read or to learn to read. Three points are important.

(1) It is clear from the facts (*a*) that non-readers are found among all degrees of intelligence; and (*b*) that the degree of failure in reading, at least among normals and high-grade defectives, shows little correlation with the degree of "general defect" commonly so called, that the defect underlying inability to read is to a certain degree specific in nature. The idea that deficiency in reading power is always an indication of a deficiency of a more general kind is, therefore, by no means supported by the psychological facts; although, of course, the condition known as "general defect" is more frequently accom-

panied by loss of power in this as in all other abilities than is normal mentality.

(2) Yet there is nothing in the results of the experiments to indicate the existence of any such region as a "visual-word" centre, the absence of or injury to which will make the visual recognition of words impossible. The defects found are not so strictly localized as such a hypothesis would demand, for the word-blind individuals reveal special difficulties in dealing with material other than words. Further, the implication of this theory that ability to read depends on the power to store up images of words has no psychological support; the recall of images is not in question.

(3) The theory that the experiments do support is that "word-blindness" is but one aspect of a more general, yet still in itself specific, defect in either the visual or auditory regions or in both. All the non-readers examined showed a reduction of the normal power in dealing with forms visually presented—especially when these forms were very like each other, their defect being shown most definitely in their failure to remember such forms. Further, certain of the non-readers showed corresponding defects in the auditory region—they could not readily discriminate or retain similar sounds. Some of the worst cases had defects of both kinds. Taking the group of non-readers as a whole, however, no correlation could be found between auditory and visual ability, i.e., these defects appeared also to be specific, although occasionally found together in one subject.

Certain of the experiments indicate that the cause of the failure to associate, as well as to retain, sounds and forms, lies to some degree in this primary disability of the auditory or visual regions, resulting as it does in the failure of the forms or sounds presented to gain any meaning. Whether there is also a failure in primary retention, declaring itself in the failure to form memory images to a normal degree, is not known. An inquiry into the use of imagery, depending as it does on the introspection of the subject, is difficult with defective children. Many of them used visual imagery in other matters, but they may, nevertheless, have been unable to visualize forms which had no meaning for them.

A PROPRIOCEPTIVE REFLEX AND CLONUS AS STUDIED IN THE SPINAL FROG.

BY KANSHI SASSA.

(From the Physiological Laboratory, Oxford.)

It is somewhat remarkable that although the frog so commonly has furnished the material for laboratory observations on muscular and lower nervous reactions, and although the experimental work upon the knee-jerk and other tendon reflexes has been so extensive, the literature of this latter subject contains, so far as I can find, but one reference to tendon reflexes in the frog. That reference is by Langelaan [3] (1901), who mentions the knee-jerk as being obtainable in a lively frog. I imagine that the reason for the general absence of reference to these reflexes in the frog is that their elicitability in that animal is quite exceptional. Rollett [5] reported that no reflex is evoked from the sterno-radialis tendon of the spinal frog in any way in spite of the existence in it of abundant nerve organs. I have myself never obtained unequivocal evidence of the knee-jerk in the frog. I find, however, that there is a reflex of the proprioceptive kind quite commonly elicitable in the spinal frog, and especially well marked in the autumn and early winter, when the animals are more excitable reflexly and in good condition. This is a proprioceptive reflex not of an extensor but of a flexor muscle, semitendinosus, a flexor of the knee. In the decapitated preparation a quite slight pull on the tendon of semitendinosus evokes a well-marked contraction of the muscle.

I have taken graphic records of this reaction in the following way. The isolated tendon of semitendinosus was carefully prepared and all other tendons inserted round the knee-joint were detached. The semitendinosus tendon was attached by a thread to the vertical short-arm of a light crank lever myograph. To elicit the reflex the horizontal long-arm of the lever, carrying the writing point, was lifted by the finger so as to slacken the muscle. Then, on removing this support, the fall of the axially weighted lever gave a sudden pull to the muscle and this slight pull evoked the reflex contraction. The tension of the pull amounted only to a few grammes. Section of the roots of the sciatic

nerve immediately annulled the reaction. A slight tap upon the thread stretching the semitendinosus was invariably followed in the spinal preparation by the contraction of the muscle. No reflex contractions could be elicited by similar means either in the quadriceps femoris or in the gastrocnemius. Nor in these latter muscles (extensors) was there evidence of tone, but in spinal semitendinosus there was plenty of tone, especially in the autumn and early winter specimens. My experiments were made during the winter months of 1920.

The reflex contraction elicited by the sudden slight stretch resembled in the form of its myogram a single twitch (fig. 2). Its myogram was strikingly different in course and duration from that of the reflex contraction evoked in the same muscle by a single moderate break-shock applied to an ipsilateral afferent nerve ; the latter was far stronger and longer (fig. 1). The contraction of the simple proprioceptive reflex

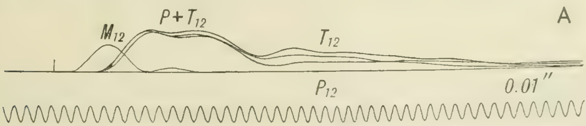


FIG. 1.—Reflex contractions of semitendinosus by single maximal break-shock. Stimuli applied to tibial (T), peroneal (P), and both nerves together (P and T). M is the direct maximal muscle twitch by stimulation of divided roots of sciatic. Numbers indicate distance between primary and secondary coils.

occupied about 0·25 second, which is little longer than that of the simple twitch at the same temperature and season. Thus in one comparison the proprioceptive reflex contraction of the muscle lasted 0·25 to 0·27 second, and the motor twitch of the muscle to a break-shock lasted 0·23 second. The whole form of the curve was very similar in the two. In this respect it seems to resemble another reflex of proprioceptive kind, namely, the knee-jerk. Jolly [2] and recently W. Salomonson [6] have examined the electrical variation of the knee-jerk, and report that the contraction of the muscle—extensor in their case—is accompanied by a simple diphasic variation. As to the latency of the reflex, owing to the mode of excitation not more can be said than that it was not longer than 0·04 second.

When the thread tied to the tendon was gently pulled by the fingers it could be noticed that the reflex response was frequently not one contraction but a short series of successive contractions. The same result could be obtained by increasing the height from which the lever fell for eliciting the reflex, thus increasing the momentum employed in

the stimulus (fig. 2). The reflex response was then a short rhythmic series, each succeeding one smaller than that just preceding it. Sometimes this multiple form of the reflex passes over into a long, lasting clonus. In the graphic records of this clonus the first contractions were of greater amplitude and a little slower period than were the rest, which

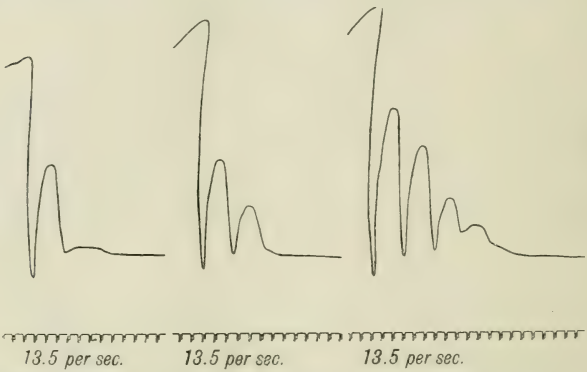


FIG. 2.—Three stretch reflexes from a semitendinosus preparation (showing increase in response with increase of stimulus).

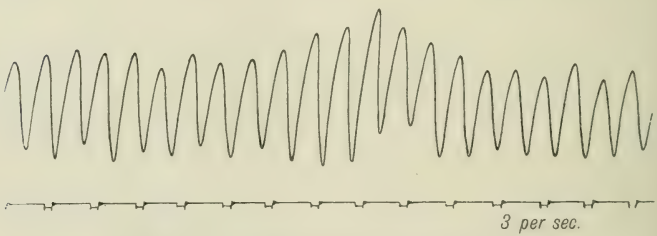


FIG. 3.—Clonus in the semitendinosus of the frog.

were often almost as regular as those of a tuning-fork. The rate varied between 4 to 6 a second; its average was 4·5 a second (fig. 3). The clonus lasted in some cases even for a couple of minutes. It would seem, therefore, that when the momentum of the falling stimulus is suitably adjusted so as to give a repetition of the required tension at suitable speed, the reflex response is elicitable in immediate succession over and over again. But the rate of this repetition is slower in the frog than

in the vastocrureus (knee-extensor) of the cat (Viets [8]), 12 to 17 per second. It is also slower than the ankle clonus (gastrocnemius) of man, which varies between 8 to 10 per second (Waller [9]), and 13·5 to 14 per second (MacWilliam [4]). This proprioceptive stretch-reflex obtainable from the semitendinosus of the spinal frog clearly resembles the reflex of similar type studied in this laboratory by Asayama [1] in the tibialis anticus of the cat. The readiness and relative regularity with which the proprioceptive stretch-reflex is, as the observations show, obtainable from a hind-limb flexor muscle of the spinal frog, but not from the hind-limb extensor muscles, may be connected with the distribution of the postural spinal tone in the two sets of muscles, this tone being in the spinal frog, as Sherrington [7] has pointed out, marked in the flexors but very low in the extensors, in accord with the habitual posture, squatting. The elicitability of the proprioceptive reflex seemed to be good when the spinal tone was well developed.

Conclusions.

A slight quick pull on the tendon of the knee flexor, semitendinosus, in the spinal frog evokes a twitch-like reflex contraction.

This is a reflex contraction, but it is strikingly different in course and duration from the reflex contraction of the same muscle obtained in response to a single break-shock applied to an afferent nerve.

Often this reflex is multiple, i.e., repeats itself under a suitable stretch stimulus. This multiple form of the reflex sometimes passes over into a clonus of long continuance and a rate of about 4·5 per second. This proprioceptive reflex appears to be favoured by the reflex postural tone which in the spinal frog is present in the semitendinosus but not in the extensors (quadriceps, gastrocnemius) ; from these latter no stretch reflex or clonus was found elicitable.

References.

[1] Asayama, C. *Quart. Journ. Exper. Physiol.*, 1916, **9**, 265.
[2] Jolly, W. A. *Quart. Journ. Exper. Physiol.*, 1911, **4**, 67.
[3] Langelaan, J. W. *Arch. f. Physiol.*, 1905, **107**, 94.
[4] MacWilliam, A. *Brit. Med. Journ.*, 1901, **2**, 1595.
[5] Rollet, A. *Sitzungsber. d. Kaiserl. Akad. d. Wissensch. Wien*, 1876, **70**, III Abt., 34.
[6] Salomonson, J. Wertheim. *Brain*, 1920, **43**, 369.
[7] Sherrington, C. S. *Brain*, 1915, **38**, 191.
[8] Viets, H. *Brain*, 1920, **43**, 269.
[9] Waller, A. *Brain*, 1880-81, **3**, 179.

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DELIVERED BY

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I feel that I cannot adequately express my appreciation of the honour of being elected to a chair which in the past has been occupied not only by many distinguished men of science, but also by some whose names are indelibly imprinted upon the history of medicine. That a surgeon should be chosen to fill this position is something more than a personal honour; it is a compliment to that branch of practice in which he is engaged, implying as it does a recognition of the fact that surgery has come to occupy a not unimportant place in neurology.

In those affections of the nervous system to which surgical measures are applicable, the physician is chiefly concerned with diagnosis, whilst the surgeon must pay particular attention to the technical problems which operative treatment presents. Neither is fitted by training or experience to perform in these respects the functions of the other, and I do not doubt that if a combined physician-surgeon could be evolved, he would evince the sterility common to hybrids.

I take the opportunity of touching upon this subject in view of the well-known opinion held by Harvey Cushing that the surgeon who concerns himself with operating upon the nervous system should be his own neurologist. However attractive this may be in theory, it can never, to my mind, attain practical realization, for just as a wide knowledge of general medicine is essential to the neurologist, so is an intimate acquaintance with general surgery desirable for one who

takes seriously that part of general surgery which concerns itself with the nervous system. The acquisition of this familiarity with all branches of surgery leaves but scant time for the laborious and exacting work, both at the bedside and in the laboratory, which the training of a neurologist demands. I hold that the nervous system is not a suitable field for surgical specialism in the more restricted sense of the term.

The co-operation of neurologist and surgeon is not only of value in the diagnosis and treatment of certain diseases of the nervous system, but it also affords opportunities for the study of what Moynihan has called the Pathology of the Living. Neurology has already gained much from observations made in the operating theatre, and far more may be confidently expected in the future. The mere exposure of the cortex, the removal of tumours, section of nerve roots and other manipulations reveal phenomena which can be observed neither at the bedside nor by post-mortem examination. In more senses than one the operating theatre may be made a link between the ward and the post-mortem room. When a physician fails to witness the operations which are performed upon his patients, he not only neglects a fruitful source of information for his own future guidance, but he also deprives the surgeon of the very great advantages which accrue from consultation upon questions which may arise during the course of an operation.

In his Presidential Address to the Neurological Society in 1902, Herbert Page said: "I venture to take up the position that surgery may be even bolder than it has hitherto been in endeavouring to minimize and remove some of the causes which seem to be at the root of the later consequences of severe head injury." Among these later consequences the occurrence of epileptiform seizures stands out prominently, more so than ever before by reason of the large number of sufferers from epilepsy resulting from cranio-cerebral wounds.

Having had to deal with many such patients during the past three or four years, I have been increasingly impressed by the feeling that surgical procedures for the treatment of epilepsy are greatly hampered by the obscurity which still exists as to the essential factors that underlie convulsive attacks in general.

James Taylor has recently reminded us that in regarding epilepsy as a disease, there is some danger of losing sight of its purely symptomatic aspect, and has emphasized the desirability of searching in every case for the physical basis which forms the starting point of the convulsive seizures. He contends that the attempt to enmesh epilepsy in the net of psycho-analysis represents a definitely retrograde move-

ment, whilst any real step forward in the elucidation of the nature of "ordinary epilepsy" is to be made from a study of Jacksonian attacks.

Gowers said that a tumour or spicule of bone could "so train the whole brain into a habit of discharge that the attacks differ little from those of idiopathic epilepsy." Instead, however, of merely seeking to reconcile the convulsive seizures of known focal origin with those of idiopathic epilepsy, we should rather approach the problem from the opposite point of view, realizing that in idiopathic epilepsy we have phenomena unassociated with any obvious cause, which may yet be in all respects identical with those which occur in connexion with gross lesions, from the damp squib effort of *petit mal* to the full pyrotechnic display of a major attack. Further, we must remember that between ordinary epileptic fits and focal fits associated with visible lesions there exists a bridge in the form of attacks of a Jacksonian character in which no gross lesion appears to exist.

Surgery would be in a better position to play its part in the treatment of epilepsy, whether or not of focal origin, if we knew more of the factors underlying the phenomena of a fit; and if we could recognize any essential precipitating cause common to all forms.

I would not presume to criticize the various views as to the pathogenesis of epilepsy which have been advanced in the past, or which are at present entertained. I should like, however, very briefly to examine what is known as the vascular hypothesis, because during the frequent opportunities which I have had of viewing the living brain, under both normal and abnormal conditions, I have been greatly impressed by the striking rapidity with which visible circulatory changes may occur in response to various disturbing causes. I cannot help feeling that sufficient importance is not attached to the possible effects upon cerebral function of what may perhaps be called minor and transitory circulatory disturbances. It is not upon the congestion or anæmia of the brain as a whole that I would lay stress, but upon the local variations and abnormalities which can be observed in connection with tumours and various traumatic lesions. It seems to me that the only local influence which an operation can have upon an epileptogenous area, short of excising that portion of the cortex, must be exercised through the medium of the local circulation. If there is a fundamental precipitating cause for all convulsive attacks, any hypothesis which does not take into account the pathogenesis of fits originating in the neighbourhood of a gross lesion must fail to touch that essential point.

Gowers, discussing the pathology of idiopathic epilepsy in 1900,

wrote: "The conceptions of epilepsy which have been current during the last forty or fifty years . . . are not much more instructive than the old demoniacal pathology which gave the disease its name." One such conception he specified, namely, that "the epileptic fit was the result of a spasm of the arteries of the brain," and went on to remark that this notion had disappeared absolutely before the simple but conclusive facts which connected the manifestations of the disease with a morbid state of the cortex. It is noteworthy, however, that, some years later, in the "Borderland of Epilepsy," he admitted the possibility of cerebral vaso-constriction in relation to vaso-vagal attacks. But the view that the phenomena of epilepsy result from disordered cortical function does not by any means necessitate relinquishing all belief in an underlying vascular disturbance.

On the contrary, both the "morbid state of the cortex" postulated by Gowers, and the "spasm of the arteries of the brain" denied by him, may conceivably be concerned together.

The whole subject of the relation of disorders of the cerebral circulation to convulsive attacks, as well as to loss of consciousness and other clinical manifestations, was fully discussed by A. E. Russell in his Goulstonian Lectures in 1909. To these lectures I have long been indebted for suggestions and references bearing upon the very interesting and important question of the relationship between gross cerebral lesions and epilepsy. For it is obvious that in operating upon epileptics, and in choosing cases for operation, we should like to have a clear idea of the purpose of the operation, based upon some reasonable conception of that relationship. The surgeon who merely removes a disc of bone from the cranium of an epileptic patient places himself on a level with the practitioner of the Stone Age, the marks of whose handiwork upon the skulls, probably of the epileptic or the insane, are to be seen in museums of ethnology.

The thesis which Russell developed from a mass of clinical evidence was that the common factor underlying many disturbances of cerebral function is to be found in some disorder of the cerebral circulation. It is interesting to note, in view of the trend of recent research, that as long as twelve years ago he hinted that, so far as the causation of epilepsy at least is concerned, inquiry into the processes, both nervous and chemical, by which the cardiovascular apparatus is governed, might afford a valuable clue. He further drew attention to a similarity between experimental decerebrate rigidity and the tonic spasms occasionally observed in cases of sudden complete arrest of the cerebral circulation.

In most writings upon epilepsy such expressions as "sudden discharge of nerve force," "sudden liberation of energy," and "explosion of nervous energy" are commonly found. Such terms, if they relate to the cortex alone, can only mean that the fit is to be looked upon as a manifestation of excessive cortical activity. It is impossible, however, to regard loss of consciousness, one of the most sudden and striking events, and often the only recognizable event in an epileptic fit, as a manifestation of the discharge of nervous energy.

A sudden failure of the cerebral circulation is inevitably followed by loss of consciousness, and we know that it can also produce convulsive movements, as seen in the attacks associated with the condition known as "heart block." It is inconceivable that a sudden arrest of the cortical circulation can, at one and the same time, bring about both a suppression and an augmentation of function. When loss of consciousness and convulsive movements result from the same cause, we can only believe that those movements point to a suspension and not a stimulation of cortical activity. The tonic stage of muscular contraction appears to indicate, not a discharge of nervous energy from the cortex, but the liberation of a lower level motor mechanism from cortical control. In a word, the unconscious rigid epileptic is, for the moment, a decerebrate man.

In a recent paper Kinnier Wilson has brought forward clinical evidence to show that decerebrate rigidity, both with and without the occurrence of tonic fits, occurs in man as the result of lesions which interrupt the cortico-spinal path, and that such rigidity is strictly comparable with that which obtains in animals when the mesencephalon has been transected. He points out that lesions which have not the completeness of the experimental section find expression in rigidities of limited scope, and that even functional interruption of cortical control may be manifested by a similar sequence of events. It requires no great effort of imagination to suppose that cortical control may be suddenly and momentarily cut off by a sudden and transient disorder of the cortical circulation, and cause, according to the extent and duration of the circulatory disturbance, varying degrees of decerebrate rigidity.

Whilst a state of momentary decerebration may be held to explain the tonic stage of an epileptic fit, it is not so easy to account for the clonic movements. Kinnier Wilson, in the paper alluded to, having characterized the tonic fits observed in the cases which he was describing as "attacks of decerebration," remarks that the absence of

any clonic movement is of much significance. He takes it to indicate that tonic and clonic movements have different origins, and are the expression of the activity of different motor mechanism, the one being characterized by static, and the other by phasic activity.

In a few recorded cases an epileptic fit has occurred during actual examination of the heart or radial pulse. The tonic stage has coincided with the sudden arrest of the circulation, whilst the clonic stage has commenced with the return of the circulation. In cases such as Russell's where sudden and permanent arrest of the circulation was immediately followed by tonic spasm, no clonic movements were observed.

It can hardly be doubted that the clonic movements which follow the tonic stage of a general epileptic fit are of the same character and depend upon the same motor mechanism as those of a Jacksonian fit which is accompanied neither by a tonic stage nor by an initial loss of consciousness. They exhibit the phasic character of movements associated with cortical activity, and resemble both those which can be evoked by faradic stimulation of the cortex, and those of a Jacksonian fit dependent upon a cortical lesion.

Many years ago, Long Fox, who attributed the tonic spasm of an epileptic fit to cerebral vaso-constriction, ascribed the clonic movements to a "gradual yielding of the vasomotor constriction, allowing at first more blood to enter the arteries than during the period of tonic spasm, but yet far less than is necessary for controlled movement or for rest."

That the cerebral circulation is under direct vasomotor control was long denied, chiefly owing to the work of Leonard Hill, who, believing the variations to be entirely passive, summed up his views in the paradox that "the cerebral vasomotor nerves lie in the splanchnic area." Although the cerebral circulation differs from the circulation elsewhere in many ways, and particularly in its passive dependence upon external circulatory changes, it is now generally admitted to be also subject to active vasomotor control, although the final demonstration of this by direct physiological experiment is still lacking.

Harvey Cushing noted that epinephrin will blanch the pial vessels over the area of its application, and that faradic stimulation will do the same. Brodie and Ferrier found that on injecting adrenalin into the basilar artery of the removed brain the outflow from the torn sinuses was diminished or completely stopped according to the quantity injected.

During the course of an experiment which Professor Sherrington kindly gave me the opportunity of witnessing, I was struck by the fact that when a faradic stimulus of sufficient strength to evoke epileptiform movements was applied to the cortex, the pia-mater could be seen to blanch, and it naturally occurred to me to wonder whether epileptiform attacks starting from a traumatic lesion may not be accompanied or even initiated by a similar blanching of the cortex.

The evidence in favour of vasomotor activity in the brain is abundant, and it seems incredible that pathological variations in the cerebral circulation should not be associated with definite clinical manifestations. The characteristic rapidity and suddenness of vascular changes, and their characteristic tendency to recurrence, would allow of recurrent phases of disordered cerebral function, and on this hypothesis many of the phenomena of epilepsy can be fully explained.

The cerebral cortex has been raked over and over again by successive generations of neuro-histologists without yielding up the secret of the cause of idiopathic epilepsy, and it may be that the search, transferred from this barren field to that of the autonomic nervous system, will prove more fruitful.

Before discussing the part played by gross lesions in focal epilepsy, it is necessary to refer briefly to the effects of electrical stimulation of the cortex.

In man the effects of such stimulation appear to resemble closely those obtained from anthropoids, but as the recorded observations are comparatively few, and not primarily experimental, this need not be referred to further, except for one important point. Fedor Krause, in 1911, had the opportunity of stimulating the cortex of a patient who was the subject of Jacksonian epilepsy without gross visible lesion. He found that faradization of the epileptic zone elicited fits, whereas from the rest of the motor cortex only the corresponding movements were obtained, identical currents being used, and the duration of the stimuli being the same in each case.

In this connection an interesting and probably unique experience recorded by Sherrington is of considerable importance. Having by chance found amongst his laboratory animals a monkey which was the subject of epileptiform attacks, he took the opportunity of investigating the effect of cortical stimulation. The spontaneous attacks had begun in the left angle of the mouth (or perhaps in the tongue), and had spread to the rest of the face, neck, left arm, left leg, and then to the right limbs. Attacks could be induced by the taking of a large morsel

of food into the mouth. A minimal faradic stimulus applied to the tongue area of the right hemisphere provoked a tongue movement which almost immediately became clonic and epileptoid, meaning by the latter that it continued as a series of movements after the faradic stimulus of the cortex had been withdrawn. If the stimulus were continued for a few seconds the epileptoid movement spread from the tongue, and would occasionally involve all the facial muscles on the left side, but it never spread beyond the face. In the left hemisphere the tongue area yielded epileptic discharges easily, though not so readily as did the tongue area of the right cortex, nor did the movements spread. Sherrington remarks that it must be remembered that limited epileptiform discharge is, in the monkey, usually obtained by prolonged or quickly repeated faradic stimulations of almost any point in the motor cortex, and that therefore the result observed with the tongue area of this animal was of a quantitative rather than a qualitative nature. Further, that the exceptional readiness with which tongue epilepsy could be evoked was remarkable because, in his experience, the tongue area is not one from which epilepsy is usually easily elicited. The results of cortical stimulation in this epileptic monkey fall into line with those obtained by Krause in his epileptic human subject.

We require, however, an explanation of the fact that at least in the highest apes (and probably in man, to judge from Krause's observations) electrical stimulation is incapable of provoking a Jacksonian fit of any magnitude. Even in Sherrington's epileptic monkey: "By no persistence of the faradization could the epileptoid movement be made to extend beyond the face. It never, unlike the natural seizures observed in the animal, spread to the neck or limbs even of the same side, let alone the opposite." We know that in experimental work the depth of the narcosis affects the results in a marked degree, and it is possible that it is an important factor in determining the differences noted. It may be, too, that higher control, whether consciously exerted or not, accounts for differences in the extent to which the cortex responds to the stimulus of a focal lesion. It is a matter of common observation that patients can often arrest the spread of clonic spasms by holding firmly the affected limb at the commencement of a fit, by which manœuvre controlling influences are doubtless directed to the cortex involved, so as to neutralize the effect of the abnormal stimulation.

Horsley and Schäfer many years ago demonstrated the fact that the excitability of the cortex is increased by repeated stimulation, so that a weaker current will evoke a spread. This was confirmed and ampli-

fied by Leyton and Sherrington, who, in the course of their work upon the cerebral cortex, elicited many additional facts bearing upon focal epilepsy. Great variations were observed among individual animals in the ease with which a Jacksonian "march" could be provoked by the faradization of a cortical motor point. Not only is the threshold of excitability lowered by faradization, but the area from which a given movement can be obtained may be gradually extended. Thus after provoking movements of the lips, for example, by stimulating a cortical point just in front of the central sulcus, the same movement may be elicited by faradization of a previously inexcitable point more anteriorly situated. Further, faradization of the post-central cortex may facilitate the elicitation of movement from certain points in the precentral cortex at about the same horizontal level.

In this connection the following experience is of interest. Working with Dr. Gordon Holmes I had the opportunity of observing the effect of faradic stimulation of the cortex in a patient who for several years had been the subject of frequent focal fits resulting from a traumatic lesion. This lesion involved an area of the cortex, some 4 cm. in diameter, both behind and in front of the Rolandic sulcus, at about the level of the temporal crest. The stimulation was carried out in order if possible to identify the point of origin of the fits, and the technique employed was the same as that used by Sherrington. For obvious reasons the observations could not be made with the completeness of a physiological experiment, but there was no doubt as to the readiness with which epileptoid movements could be evoked from many points, some of which were far distant from the cortical area of normal representation.

Leyton and Sherrington have named the movements obtained by electrical excitation of the cortex "fractional" movements, and remark that they are in themselves co-ordinate and seem to form parts of complex purposive acts.

Electrical stimulation of the cortex, even when carried out with every possible refinement of technique, must be the grossest caricature of the natural stimuli which provoke purposive movements. Yet such electrical stimuli applied to the precentral cortex in man can be made to evoke definite movements, limited in extent and bearing a striking resemblance to simple natural movements. Although, as Ferrier first pointed out, these movements exhibit purposive co-ordination, they are not purposive movements, and in this respect resemble those of mild epileptiform attacks.

A Jacksonian fit is merely a series of fractional movements, devoid of purpose, and determined in point of sequence only by the relative anatomical position of the cortical areas in which they are represented. In other words, a Jacksonian fit is made up of a series of fractional acts, which, although in themselves co-ordinate, lack the mutual co-ordination of natural purposive acts. This mutual incoordination of fractional acts is illustrated by the biting of the tongue, due to the unnatural sequence of tongue protrusion and jaw closure. In this respect the movements of epilepsy resemble the effects provoked by electrical stimulation, which even when so applied as to evoke a "march" or sequence of movements, does not appear ever to call into action the peculiar synthetizing function of the cortex defined by Sherrington. No series of fractional movements so combined as to bring about a recognizable purposive act, even of a simple character, has, so far as I am able to ascertain, been recorded as resulting from electrical stimulation of any part of the cortex. In fact no more intelligent movements can be obtained from the cortex than from the mid-brain; they differ in character rather than in purpose, the one set taking the rhythmical form characteristic of movements obtained from the cortex, and the other lacking that particular nature. It seems to be not impossible therefore that the disturbance of cortical function, whether caused by experimental stimulation or by a gross pathological lesion, may, in part at least, be due to a lowering of the normal inhibition, and that the movements which result may be of the nature of release phenomena. On such a conception the epilepsy resulting from pathological lesions can be brought into line with that which can be induced experimentally.

The association between gunshot injuries of the brain and the development of fits is so obviously a case of cause and effect that I will refer to them before touching on the subject of fits associated with morbid lesions, and on those of idiopathic epilepsy.

The local morbid conditions underlying the fits which result from gunshot wounds of the head fall into three main groups :—

(1) Recent lesions such as local contusion associated with small hæmorrhages and œdema; or the more gross disruptive effects of a penetrating wound.

(2) Inflammatory lesions due to recrudescent sepsis, where a latent infection becomes active and gives rise to an area of softening, or to an actual abscess.

(3) Cicatrices binding the scalp to the brain and membranes through a cranial defect.

As regards the first two groups, the early fits, namely those which occur in the first few days after the injury, as well as those connected with recrudescent sepsis, are clearly associated with vascular disturbances due to direct damage to blood-vessels, thrombosis, inflammation and œdema; they continue only during the period of acute circulatory disorder, and cease when that period comes to an end.

The third group, with cicatrices binding the scalp to the damaged brain, is by far the most important, and it is to the cases included in this category that I wish to draw particular attention.

A year ago the Re-survey Boards of the Ministry of Pensions had, in the previous twelve months, examined more than 25,000 old cases of gunshot wound of the head. Excluding those examined more than once during that period, the number of individuals was approximately 18,000, amongst whom nearly 800 (or 4½ per cent.) were the subjects of epileptic fits. A very large number of such patients present a cranial defect through which the scalp adheres to the membranes and the damaged brain. Penetration of the dura at the time of the wound entailed not only gross direct damage to the underlying brain and its vessels, but also invasion of the injured tissues by micro-organisms. The consequent inflammation resulted in further destruction of nerve tissue as well as a greater disturbance of the vascular supply, both from thrombosis at the time and from strangulation of vessels later by fibrous tissue, the amount of which would to a large extent be proportional to the intensity and duration of the preceding inflammation. Further, the penetrating wound is inevitably followed by adhesions between the surface of the brain and the scalp.

It may reasonably be assumed that the rich blood supply of the normal cortex provides a wide margin of safety against accidental variations, whilst the relatively avascular scarred cortex possesses a smaller margin of nutritional safety. In these circumstances local circulatory disturbances would be likely to arise from accidental causes. One such cause which I believe to be of the very greatest importance, depends upon the fixation of the brain at the point of damage to the overlying membranes and scalp, and is occasioned by the cerebral movements.

There is no doubt that the normal brain obeys the law of gravity, and alters its position with varying positions of the head. Every organ of the body possesses a degree of mobility proportional to the extent to which it is covered by a serous membrane separating it from the wall of the cavity which contains it. The serous membranes of the thoracic

and abdominal cavities, as well as those which line the joints and the tendon sheaths, result from and facilitate movement. The brain is no exception ; the serous cavity lying between the dura and the arachnoid can fulfil no other purpose ; the intense pain occasioned by any movement of the head in meningitis is analogous to that of pleurisy. Whenever the skull and dura are widely opened during an operation, movements of the brain can be demonstrated by altering the position of the head, and although their extent is probably exaggerated by reason of the different physical conditions which obtain in the open as compared with the closed skull, there is no reason to suppose that all movement is absent when the skull is whole. Indeed J. Luys has demonstrated the fact of cerebral mobility by means of frozen sections, and Gavoy has, by means of a specially designed cerebral kinesiometer, been able to measure excursions of from three to nine millimetres.

When a brain, attached to the scalp by adhesions, attempts to move in response to a change of posture, it is prevented from doing so from a pull at the point of anchorage ; this pull either mechanically or, possibly, by causing a reflex vasoconstriction, may well produce a momentary local anæmia in the damaged brain, and so initiate a fit.

Seeing, however, that only a very small number of patients with anchored brain develop epilepsy, it is obvious that the local lesion is but one of the links in the ætiological chain. Clearly a given stimulus is not effective for all brains, and it is necessary to assume in those patients who do develop fits a tendency to epilepsy resulting from what is vaguely called a relatively low degree of stability of the nervous tissue.

I have been fortunate enough to encounter a case which illustrates this important point remarkably well. A man, 35 years old, had at the age of 15 been accidentally wounded in the forehead by a revolver bullet. He was operated upon shortly after the accident, and was in hospital about fifteen weeks. He afterwards remained perfectly well in every way, joined the Motor Transport Service in 1915, and was sent to France where he served until 1919. He received no wound or other injury, but was often under shell fire and bombing. His first fit occurred after six months' service, and between that time and the date of his admission to hospital in 1920 he had about two dozen fits, all of which occurred during sleep, except two. The fits associated with frontal injuries of this character not infrequently occur during sleep, and they are, if the explanation which I have suggested is correct, to be attributed to the sudden changes of posture which may occur during sleep. This patient, in the two attacks which occurred in the daytime, fell unconscious,

without warning, and remained so for some minutes. He did not bite the tongue nor urinate; he suffered neither from headaches nor giddiness. There was a small irregular circular opening in the right frontal bone, into which the brain bulged on stooping forward. No abnormal neurological signs were detected.

The interpretation of this case seems to be that, although for twenty years this man had had a cranial defect through which the brain was anchored to the scalp, the local lesion had been insufficient to cause a fit until the nervous stability had been lowered by the stress of warfare. In May, 1920, an operation was performed. The scalp was dissected free from the dura, and the dura was separated from the bone for an inch or more beyond the edge of the opening. A thin sheet of celluloid was placed over the dura, extending some distance between it and the inner surface of the skull. A thicker sheet of celluloid was used to close the bony opening. Some ten weeks later he had a fit, whilst asleep, and since then no further fit has occurred.

In cases of this class the aim of an operation is twofold, first to ensure a permanent separation of the brain from the surface at the point of damage, so as to restore as far as possible its mobility; and secondly to close the bony opening so as to restore the physical conditions under which the cerebral circulation normally works. Both of these objects can be attained by the use of celluloid, the technique of which procedure will I hope form the subject of a future communication.

I and my assistants at the Tooting Pensions Hospital have now operated upon more than 200 cases by this method. About 120 of these patients suffered from fits. The time has not yet arrived for passing a judgment upon the results, but I may say that so far they are remarkably promising, even in some cases where the fits had occurred over a long period. Nor is it possible to estimate fairly the extent to which the beneficial results are due to the other treatment carried out at the same time, for in all cases the patients have been kept in hospital for from two to six months after the operation, and treated regularly with small doses of bromide.

It seems probable, to judge from our experience, that better results may be expected in cases where the fits have been infrequent, and have not occurred over a long period of time. Nevertheless, satisfactory results are not impossible even in long-standing cases, as shown by the following example:—

A man of 21 had, when 7 years of age, received a severe cranio-

cerebral injury in the left parietal region, which resulted in gross hemiparesis. A large area of bone had been removed, and the damaged brain had become adherent to the scalp. The brain pulled upon the scalp, which became concave when the patient lay upon his right side; the brain bulged when the head was inclined to the left.

Eleven years after the accident he began to suffer from fits, the origin of which was attributed to a blow upon the head in the region of the cranial defect. When admitted to St. Thomas's Hospital he had had fits for three years, for the first year about once a fortnight, for the second year about three times a week, and for the third year about once a month. On one occasion he had twenty-five fits in a single day. He was operated upon by the celluloid method and carefully treated for several months afterwards. Two years have now elapsed since the operation, during which time he has had only two fits. He has taken no drugs for a year, has improved greatly in general health, and is able to do some light work.

The claim of surgery to participate in the treatment of fits resulting from injuries to the skull and brain may fairly be regarded as well established, not only on the ground that a reasonable explanation exists of the mechanism by which they are evoked, but also because we now possess a good deal of evidence to show that the fits can in many cases be either abolished, or very considerably reduced in number and severity, by an operation which succeeds in removing or at least modifying, the local exciting cause. This evidence, now in course of preparation, I purpose bringing forward in the near future.

Leaving now the subject of epileptiform attacks due to gross injuries, we must approach the question as to what contribution surgery has to offer towards the treatment of fits other than those of traumatic origin, whether associated or not with local lesions of the brain. For the purpose of examining this question, I would divide these cases into three groups:—

The *first group* comprises all the cases in which there exists a lesion both lending itself to accurate localization and allowing of a reasonable surmise as to its nature. Many tumours, both innocent and malignant, as well as abscesses, cysts, tuberculomata and gummata, can be included in this category. Whilst any of these lesions may constitute the starting point of an epileptic attack, it must not be forgotton that there is no gross lesion, whether traumatic or otherwise, which necessarily causes fits. On the contrary, the proportion of patients with any such lesion who develop fits is small. As already mentioned, the incidence of

epilepsy amongst pensioners with old gunshot wounds of the brain does not at the present time reach 5 per cent.

The frequency, however, with which fits are associated with tumour of the brain is perhaps somewhat striking. I have recently looked through the notes of 270 cases of brain tumour, exclusive of cerebellar and pituitary cases, upon which I have operated. I find that no fewer than eighty-two patients, or 30 per cent., exhibited fits of a focal character. Grouping regionally the tumours associated with fits, I find that exactly half were situated precentrally, and of the remaining 41 22 were post-central, 12 temporal and 7 occipital. Of the whole 82 tumours, 65 were malignant and 17 were endotheliomata.

In no fewer than 40 per cent. the fit was the first symptom of which the patient complained. The fit was the first symptom in a rather larger proportion of the endotheliomata than of the gliomata, but the difference is not, in my opinion, sufficiently striking to be of any great value in diagnosis.

There can be no doubt, I think, that the nearer the tumour, or the surrounding area of cerebral softening, approaches the central sulcus, the more likely are fits to occur.

The exact nature of the stimulus by which a tumour or other gross lesion elicits evidence of cortical disturbance, whether of a motor or of a sensory character, is not easy to define. To say that a lesion "irritates" the brain merely creates fresh difficulties by raising the question of the exact meaning of irritation. Used in its usual sense irritation implies activity, but a lesion such as a bony boss resulting from a depressed fracture cannot be regarded as an active agent. If the brain from time to time accidentally, or continually on account of its respiratory and cardiac movements, impinges upon such a bony boss, changes conducive to epilepsy might occur. A tumour might be in a sense "active" from alterations in size or tension due to variations in its blood content, or to such sudden gross change as that caused by a hæmorrhage into it. Active inflammatory changes also occur from time to time in the membranes bounding a cortical tumour, whilst naturally vascular disturbances are a prominent feature where frankly inflammatory lesions are concerned. In the immediate neighbourhood of many of these lesions the appearances are such as to suggest that rapid or sudden alterations of vascularity could readily take place, and such changes seem to me to constitute the one common factor which is capable of explaining the associated epileptiform movements, and so bringing their causation into line with that of other forms of epilepsy, whether traumatic or apparently spontaneous.

In the surgical treatment of the cases included in this first category, namely of recognizable and localizable gross lesions, the operation is naturally directed primarily to dealing with the tumour or other lesion, and the result as regards the fits, as well as other symptoms, will depend upon its nature and the completeness with which it can be removed. When a simple tumour can be completely removed, or an abscess shelled out entire with its capsule, the outlook is good. When an abscess has to be drained, fits are not so likely to be abolished, since the drainage necessitates anchorage of the brain to the surface, which is in itself, as I have endeavoured to show, provocative of epilepsy.

The *second group* comprises those cases in which fits of a definitely focal character occur, but in which the nature of the lesion, if any, cannot be ascertained without actual inspection of the brain. Operation, which is fully justified in many of these cases, must necessarily be primarily of an exploratory character. In spite of the fact, however, that the technique of cranial operations has been so greatly improved, we must not fall into the error of putting a cerebral exploration quite on a level with an abdominal exploration, or undertake it with the same confidence that, even if nothing is found, no harm will result. By such exploratory operations I have found tumours, meningeal cysts, on one occasion a nævoid angeioma, and on another occasion a small abscess, all of which were capable of being satisfactorily dealt with.

On the other hand, a wide exposure of the cortex may reveal no visible lesion of any kind; the brain may appear to be absolutely normal both to inspection and palpation. In such a case the question naturally arises as to whether that portion of the cortex in which the initial movement is represented, having been defined by electrical excitation, may be excised. The effects of cortical excision upon function are, thanks to Horsley, Sherrington, Marinesco and others, well known; but the results of such excisions in patients suffering from focal fits without visible lesion have not, even in the hands of the most expert operators, proved very encouraging.

Cushing, from an experience of some fifteen cases, writes as follows: "Some had unexpectedly resulted in cures; one or two which promised to be especially favourable owing to the circumscribed nature of the initial movement (thumb and corner of mouth) were not benefited."

Krause gives details of three cases of focal epilepsy without visible lesion in which he excised what he calls the "primary spasming centre." One patient had had no fit some months later; one eight years later only had occasional twitchings; and one died shortly after the operation in status epilepticus.

Krause attributes the failure of cortical excisions in the hands of others to the localization having been made on anatomical grounds and not by electrical excitation. He believed the resultant scars to be of no importance, and not provocative of any spasm or symptoms whatever.

It is clear that in determining the advisability of performing cortical excision the resultant disabilities would be of less account in dealing with focal epilepsy starting in parts already deprived of their function. The published cases are too few and too insufficiently recorded to afford good ground for any definite conclusions. But the subject cannot be altogether dismissed without further investigation.

The *third group*, namely that of the so-called idiopathic epilepsies, includes all the cases in which the fits can neither be ascribed to any ascertainable organic lesion, nor possess any features pointing to a definite focal origin. I have searched in vain amongst the many records of operations for epilepsy, some purely fantastic, some based upon a more or less reasonable conception of its causation, for any encouragement to pursue further the quest for an operative procedure which holds out any prospect of benefiting the sufferer from idiopathic epilepsy. It may be unwise to say that the future will reveal nothing fresh in this direction, but it is at any rate certain that, at the present time, surgery has nothing to offer in the treatment of general idiopathic epilepsy.

In some other forms of epilepsy surgery may, as I have indicated, be made to play a useful part, but no operation can alone be expected to effect a cure. I am, however, convinced that, in properly selected cases, surgical treatment can be of great value in lessening the task demanded of medicinal and other therapeutic measures. If these measures cannot be carried out rigorously over a prolonged period, then, for the good of the patient and the credit of surgery, it were better to refrain from operating.

Cynical friends have told me that operations upon the nervous system do but illustrate the truth that hope ever triumphs over experience. The surgeon must be an optimist, but his optimism need not be that of the ostrich. It should be based upon the expectation that inquiry into the causation of those diseases which he seeks to remedy will eventually furnish the clue to their successful treatment. The search may lead him into deep waters, and this must be my excuse for venturing so far out of my depth as to address a gathering of neurologists on such a subject as epilepsy.

Die vergleichende Anatomie des Nervensystems der Wirbeltiere und des Menschen. Von Dr. C. U. ARIËNS KAPPERS. SS. 624. Mit 326 Figuren und 3 farbigen Tafeln. Haarlem : Bohn.

The establishment of the Central Institute for Brain Research in Amsterdam some years ago was a welcome experiment to all who are interested in the anatomy and development of the nervous system. And it became even more welcome when it was evident that, under the leadership of Dr. Ariëns Kappers, it would devote itself largely to the study of comparative nervous anatomy. Such an institute had the special opportunity to collect a body of workers who, in addition to producing individual essays on the phylogenetic development and comparative anatomy of separate portions and structures of the brain, would, under the direction of a capable chief, slowly collate and co-ordinate the knowledge that had been hitherto acquired so that we might ultimately obtain a comprehensive view, not only of the morphology of the brain, but also of the causes and factors that have determined its form in the higher vertebrates. The desirability of this is obvious since the anatomy and the adaptation to function of many structures in the body are comprehensible only when their evolutionary development is known, and this is particularly so in the nervous system.

There has been no lack of individual contributions to the comparative anatomy of separate portions of the nervous system, particularly by Continental and American workers, but the want has become acute of a textbook that would embody all the present knowledge in an accessible form for those who are unable to follow the contributions of each worker, or to sort out their results and integrate them critically into an intelligible whole. In this statement we are not unmindful of the well-known textbooks by Edinger, who must be justly regarded as father of this science, but the last edition of his book is several years old ; and great as Edinger's achievements were, he was not, owing to his peculiar and original abilities, the most fitted person to harvest the enormous field he had sown. Smaller textbooks, as those by Herrick and Johnson, have been unquestionably useful as outlines or introductions, but their plan and compass precluded a full or adequate treatment of the subject.

That this want will be supplied by Kappers' book there can be no doubt. In the first section, which has already appeared, more than 600 pages are devoted to the histology and arrangement of nervous elements, and to the comparative anatomy of the spinal cord and the medulla oblongata. The present edition is appearing in German, but though English students may

regret that their language was not chosen, those with any knowledge of the German tongue will find no difficulty in reading Kappers' simple and lucid pages, or in following his well illustrated descriptions and his concise but always intelligible arguments.

This volume must not be, however, regarded merely as a textbook on comparative anatomy, for the human nervous system is dealt with as fully as in most books devoted to it alone. In fact its chief value to the neurologist is that the descriptions of the various structures found in the lower animals are so planned that they lead up to and simplify the study of the nervous system of man.

The first fifty pages, which contain a concise account of the histology of the nervous elements and supporting tissues, will probably afford less interest to the student than the remaining portions of this volume, though the author's critical review of many points still under discussion give it a distinct value, as well as make it a necessary prelude to the anatomical chapters.

In the next section the factors which determine the forms and connections of neurons are considered. Here we are introduced to the theory of neurobiotaxis on which Kappers has already written exclusively, but never so clearly and logically. The scope of a textbook and the opportunity it gives the author of reviewing the whole field of his experience, has enabled him to put his hypothesis in a simpler and more cogent form than when he attempted to illustrate it by some particular structure or system. And it must be admitted that, as presented in these pages, neurobiotaxis is more acceptable as a general law in the development of the nervous system than it appeared to be in some of the author's earlier papers. It is also pleasing to find that throughout the volume its application is not unduly forced, as frequently happens in the case of a newly discovered principle of such importance.

The author is not so happy in the distinction which he attempts to make between what he terms "vital" and "gnostic" functions, in the treatment of the lower levels of the nervous system at least. He uses "vital" as an alternative term to "protopathic," and "gnostic" as an equivalent of "epicritic," which in itself is an unfortunate choice, as these terms have been already employed with other meanings. Further, it seems very questionable whether such a distinction is possible in the lowest vertebrates, or in functions so primitive and elementary that they are subserved mainly by spinal and bulbar reflexes. The "vital functions" of the spinal afferent nerves are enumerated as the appreciation of pain, touch, and high and low temperatures, i.e., those perceptions to which Head has applied the term "protopathic," but a differentiation that describes gravi-statik as the "vital" (protopathic), and hearing as the "gnostic" (epicritic), function of the octavus nerve is liable to cause confusion.

The chapter on the comparative anatomy of the spinal cord affords an extraordinarily interesting review of the phylogenetic development of this segment of the nervous system from amphioxus to man. Here we can see not merely the increase in complexity of its structure as we ascend the animal

scale, but the manner in which new systems have developed within it, and its variations with the morphology and mode of life of the animal. The third chapter introduces us to the more difficult subject of the comparative anatomy of the medulla oblongata, though here too the systematic way in which the subject is treated, and the judicious use of diagrams and illustrations, make the descriptions as simple as they possibly could be. The constitution and connections of the branchial nerves of the fishes is regarded as one of the chief factors that determine the structure of the medulla in the vertebrate series, and the modifications that they or their derivates undergo in the course of the vertebrate evolution is made remarkably clear in the detailed description of their morphology in all the chief classes. This may be illustrated by the manner in which the anatomy of the afferent fibres of the facial, glossopharyngeal and vagal nerves is worked out, especially in their relation to the gustatory system. The cutaneous or somato-sensory components of these three nerves join the descending trigeminal root in the medulla, and end in the same mass of grey matter as it. In fishes their visceral and gustatory fibres enter the medulla and terminate together at the level of their entry in the viscero-sensory area. The earliest differentiation is found in the amphibians, where descending fibres of these roots first form a fasciculus solitarius; this contains viscero-sensory fibres only and has no connection with the gustatory system, as its size bears no relation to the development of the peripheral taste-organs, but corresponds to the sensory endowment of the upper alimentary and the respiratory tracts. As the non-specialized viscero-sensory fibres become segregated in the fasciculus solitarius the gustatory fibres of these three nerves pass to more dorsally and mesially placed nuclei in the floor of the fourth ventricle. The latter are identified with the nucleus intercalatus Staderini of mammals, which is regarded by Kappers as the chief lower gustatory centre. On the other hand, the grey matter, which is hypertrophied in the bony fishes into the lobus facialis and there receives the bulky afferent facial fibres supplying the taste-organs situated on the surface of the body, disappears in the higher vertebrates in which the cutaneous taste buds are no longer found.

The fourth chapter is devoted to the anatomy of the lateral-line and octavus system, and the fifth to the anatomy of the motor mechanisms of the brain-stem.

In the final chapter of this volume the reticular nuclei of the brain-stem and the inferior olives are studied in detail. In the lower fibres the reticular nuclei are discrete collections of large cells, generally related in position to the motor nuclei, but in the higher vertebrates many of them shift from this position, usually moving towards the afferent centres from which they receive impulses. The majority of their axons pass spinalwards, usually through the central longitudinal bundle. Their connections show that they are adapted to correlate impressions received from various sources and transmit them by their spinalward axons to the motor effector centres. They can be consequently regarded as co-ordinating mechanisms which collect or sort out stimuli with a common

or allied end-effect. In the higher vertebrates the reticular nuclei acquire a large proportion of smaller cells. In some instances, as in the nucleus ruber, the new cells have a cerebral connection, but most of the other reticular nuclei reserve their original function of motor co-ordinating centres of a lower level.

The inferior olives are equally ancient structures in phylogenetic history. Probably present in the most primitive fishes, they are certainly distinct structures from the selachians to man, though they undergo various modifications. The most important of these is the relatively greater growth of the main olive as compared with the accessory olives in the higher vertebrates, a change that is probably connected with the evolution of the mammalian cerebellum. All researches that have been undertaken on the olives indicate that their chief efferent connection is with the cerebellum, but we are still more or less ignorant of the sources of their afferent impulses. Kappers' suggestion that it may be mainly by collaterals from the afferent systems of the cord and medulla that they come, is interesting.

The reviewer cannot leave this volume without a word of praise for the manner in which it has been produced. The type is clear, the paper is excellent, misprints and typographical errors are rare, and the illustrations are most admirably reproduced. A very complete index, and a bibliography at the end of each chapter to the subjects treated therein, add to the value of the book.

GORDON HOLMES.

The Essentials of Mental Measurement. By Dr. WILLIAM BROWN, M.A., M.D., D.Sc. (Wilde Reader in Psychology in the University of Oxford), and Professor GODFREY H. THOMSON, D.Sc., Ph.D. (Professor of Education, Armstrong College, Newcastle-upon-Tyne). Cambridge University Press, 1921.

The publishers describe this new volume of *The Cambridge Psychological Library* as "a revised and expanded edition of Dr. Brown's original work, bearing the same title, published in 1911, together with a large amount of new material by Dr. Godfrey H. Thomson." Indeed, Dr. Brown's well known book has been more than doubled in size. Of the ten chapters in the work before us, five are entirely or almost entirely new, while two more have been expanded and altered, leaving only three substantially in the form in which they were first published a decade ago. The changes are wholly the work of Professor Thomson.

Dual in origin, the book is also dual in constitution. But it must not be supposed that the two distinguished authors are respectively responsible for the two chief ingredients. These are, firstly, an account of the principal mathematical methods that have been applied to statistics obtained by psychological and other experiments, and, secondly, a contribution to the discussion of general factors—and particularly of the "single general factor"—

in mental measurements. Especially in its former aspect, the book is of high value and fully deserves a place, not only in the "Cambridge Psychological Library," but also in the library of every University, College, or other institution where psychology is studied or applied. In its other aspect, the book is somewhat less satisfactory, although it has certainly advanced matters by calling for a reconsideration of one of the steps in the argument which Hart and Spearman employed (in 1912) for demonstrating the existence of a general factor in each of fourteen series of published tests, dating from various periods up to thirty years back.

The value of the book as an introduction to the mathematics of mental measurement has been enhanced by the addition of an excellent summary of the elementary theory of probability, including several interesting examples that are not to be found elsewhere. Another new chapter will be welcomed for the account it gives of Karl Pearson's fundamental work on frequency curves. The usefulness of the book might, however, have been further increased by including a little more information on some of the subjects treated. For example, the correlation between two variables is defined (pp. 101 and 109) in algebraic terms ($r = S(xy)/N\sigma_1\sigma_2$); and, although it is stated that "if the regression is linear, r is a measure of the degree of dependence between x and y," the simple geometrical aspect of correlation is never explained. Thus, whenever the relation between x and y is linear (as it always is in the case of two correlated variables, each of which is distributed according to the normal probability law), x and y may be regarded as the projections of *the same* variable line OP and two fixed lines, Ox and Oy respectively, while the correlation between x and y is, quite simply, the cosine of the angle between Ox and Oy. In other words, this correlation measures the average deviation in x (or y) corresponding to unit deviation in y (or x). Again, the "partial correlation" between x and y with z constant (where z is a third variable measuring the projectional OP on a third fixed line Oz) is just the cosine of the angle between the projection of Ox and Oy on a plane perpendicular to Oz. If geometrical interpretations of this kind had been taken into account there would have been no occasion for certain criticisms of details that we must make later. . . . Among other matters that might usefully have been included are Pearson's geometrical interpretations of his formulæ for correlations influenced by selection (p. 135); and a fuller account of the paper by Pearson and Filon (*Phil. Trans.*, 1898, cxci, A, pp. 229-311) of which important use is made on pp. 184 *et seq.*

But the other aspect of the book will be of more immediate interest to most readers. The authors' contribution to the theory of general ability may therefore be discussed, and perhaps criticized, at greater length than their admirable synopsis of the essential mathematics of mental measurement. It should not, however, be forgotten that the criticism that follows applies only to a comparatively small part of the book under review.

The fundamental mathematical proposition in the theory of general ability is to the effect that if the correlations between the measures of a number n

(larger than three) of qualities, each of which is distributed according to the normal probability law, form a perfect hierarchy, then the measure of each of these qualities is a vector compound of a single general factor, g (that is common to all the n qualities), and of a specific factor (that is peculiar to the particular quality in question), while g and the n specific factors are independent of each other. Mathematically expressed, the proposition is that if $q_1, q_2, \ldots q_n$ are the measures, in suitable units, of these n different qualities, and if the correlations (r_{st}, &c., where r_{st} is a correlation between q_s and q_t) satisfy the $\frac{1}{2}\, n\, (n-3)$ *independent conditions for a (perfect) hierarchy—namely that* $r_{as}/r_{at} = r_{bs}/r_{bt}$ . . . . (1)
for all different values of $a, b, \ldots s, t, \ldots$ from 1 to n inclusive—then we can write—

$$q_s = g \cos \theta_s + \xi_s \sin \theta_s. \quad (s = 1, 2, \ldots n) \ldots (2)$$

or

$$q_s = r_{sg}\, g + \sqrt{1-r_{sg}^2}\; \xi_s. \quad (s = 1, 2, \ldots n) \ldots (3)$$

where $g, \xi_1, \xi_2, \ldots \xi_n$ are $n + 1$ independent factors, each of which is distributed according to the normal probability law, and where g is *the single general factor* common to all the q's, and ξ_s is a specific factor peculiar to qs, and where r_{sg} is the correlation between q_s and g.

This definition of the single general factor is not given in the book under review; nor is the above proposition explicitly stated, although mention is made of the converse proposition—that if there is a single general factor then the conditions (1) will be satisfied. Moreover, these conditions, given on p. 165, are nowhere employed explicitly to define a (perfect) hierarchy. It can only be the absence of these definitions that renders possible such a statement as that the deduction of "The Theory of General Ability, or the Theory of Two Factors" [equation (3) above] "from the occurrence of 'hierarchical' order among the correlation coefficients is invalid" (p. 154). The statement to which the authors intended to give expression can hardly have been a denial of the certain fact that, where hierarchical order is perfect, there is a single general factor. They doubtless meant that when the hierarchical order is imperfect it is explicable otherwise than by means of a general factor that will be more or less dominant, together with group factors that will be more or less insignificant according to the degree of imperfection in the hierarchy. But their phrase, as it stands, is surely misleading.

To the same cause—lack of precise definition of the terms employed—we would attribute certain statements made in the course of the third section of Chapter VII (pp. 138 to 144). The section is concerned with three correlated variables. In such a case the number ($\frac{1}{2}\, n . \overline{n-3}$) of independent conditions that have to be satisfied in order that the correlations may form a hierarchy reduces to zero, since $n = 3$. Consequently, it is *always* possible to find a 'single general factor' common to three correlated variables. And yet (on p. 140) the authors say "there is no general factor whatever," and, again, "we cannot say with certainty whether a general factor exists or

not." General factors—that are not the single general factor just defined above—can, in fact, be found for three correlated variables in a trebly infinite number of ways; for, given any three correlated variables, q_1, q_2, q_3 (distributed according to the normal law with the same probable error), it is possible to choose, in a trebly infinite number of ways, three independent variables (also distributed according to the normal law with still the same probable error) ξ, η, ζ, such that—

$$\left.\begin{aligned} q_1 &= \lambda_1 \xi + \mu_1 \eta + \nu_1 \zeta \\ q_2 &= \lambda_2 \xi + \mu_2 \eta + \nu_2 \zeta \\ q_3 &= \lambda_3 \xi + \mu_3 \eta + \nu_3 \zeta \end{aligned}\right\} \quad \ldots\ldots\ldots (4)$$

and it is always possible to choose one of them, say ξ, so that λ_1, λ_2 and λ_3 are all finite; or, in other words, so that ξ is a general factor of q_1, q_2 and q_3. If in the example in which the book before us says, "there is no general factor whatever," we put q_1, q_2, q_3 proportional to the scores obtained by throwing n purple dice (whose score, reduced to standard deviation, is p), n green dice (reduced score, g) and n orange dice (reduced score, o), so that q_1 is the reduced score of purple and orange, q_2 of orange and green, and q_3 of green and purple, we have—

$$\left.\begin{aligned} q_1 &= \frac{1}{\sqrt{2}} p + \frac{1}{\sqrt{2}} o \\ q_2 &= \frac{1}{\sqrt{2}} g + \frac{1}{\sqrt{2}} o \\ q_3 &= \frac{1}{\sqrt{2}} g + \frac{1}{\sqrt{2}} p \end{aligned}\right\} \quad \ldots\ldots\ldots (5)$$

in which, indeed, no general factor is apparent. But it is there, none the less. For it is, of course, possible here (as in every other case) to choose independent variables, ξ, η, ζ, such that any one of them, say ξ, is a general factor. For example, we may choose q_1 itself (putting $\xi = q_1$ in equation (4)) as one of the independent variables, and the plane $\xi O \eta$ to contain Oq_2. Then equation (5) becomes—

$$\left.\begin{aligned} q_1 &= \xi \\ q_2 &= \tfrac{1}{2} \xi + \frac{\sqrt{3}}{2} \eta \\ q_3 &= \tfrac{1}{2} \xi + \frac{1}{2\sqrt{3}} \eta + \sqrt{\tfrac{2}{3}}\, \zeta \end{aligned}\right\} \quad \ldots (6)$$

revealing $\xi = \frac{1}{\sqrt{2}} (p + o)$ as a general factor of q_1, q_2 and q_3; $\eta = \frac{1}{\sqrt{6}} (2g - p + o)$ as a group factor of q_2 and q_3; and $\zeta = -\frac{1}{\sqrt{3}}(g + p - o)$ as a specific factor of q_3. And this is only one of an infinite number of ways in which q_1, q_2 and q_3 may be expressed, by means of equations (4), as linear functions of three independent factors of which one is a general factor. And yet the authors say that "we cannot say with certainty whether a general factor exists or not." They add (p. 142) that the condition that three correlated variables, representing dice scores, may have a general factor is that—

$$r_{23}^2 + r_{31}^2 + r_{12}^2 + 2\, r_{23}\, r_{31}\, r_{12}$$

shall be greater than unity. But this is really the condition that it will be possible to choose ξ in equation (4), not only so that λ_1, λ_2 and λ_3 shall all be finite, making ξ a general factor (which can always be done) but *also so that certain very special conditions*, namely—

$$\frac{\mu_2}{\mu_1} = \frac{\mu_1}{\lambda_1} \text{ or } o; \quad \frac{\mu_3}{\lambda_3} = \frac{\mu_1}{\lambda_1} \text{ or } o; \quad \frac{\nu_2}{\lambda_2} = \frac{\nu_1}{\lambda_1} \text{ or } o; \quad \text{and} \quad \frac{\nu_3}{\lambda_3} = \frac{\nu_1}{\lambda_1} \text{ or } o \quad . . . (7)$$

shall be satisfied at the same time.

Nothing is said about these very special conditions, except that "it may not be out of place to remind ourselves again that this, though true of the arrangements of dice we are considering, may not be true in the same sense of other phenomena, e.g., biological or mental phenomena" (p. 142). Yet it is these special conditions, whose natures are not examined, that cause the error here and cause further misapprehensions in Chapter X (pp. 175 *et seq.*). Their origin is to be found in the fact that, while the general expression for any one (say q_s) of n correlated variables ($q_1, q_2 \ . \ . \ . \ q_n$), each of which is distributed according to the normal law with the same probable error, in terms of N (where N is not less than n) independent variables ($x_1, x_2, \ . \ . \ ., x_N$), each of which is distributed according to the normal law with still the same probable error, is—

$$q_s = {}_sl_1\, x_1 + {}_sl_2\, x_2 + \ . \ . \ . + {}_sl_N x_N \ (s = 1, 2, \ . \ . \ . \ n) \ . \ . \ . \ (8)$$

where—

$$1 = {}_sl_1^2 + {}_sl_2^2 + \ . \ . \ . + {}_sl_N^2 \qquad (s = 1, 2, \ . \ . \ . \ n) \ . \ . \ . \ (9)$$

the expression for q_s when $x_1 \ . \ . \ . \ x_N$ represent dice scores is—

$$q_s = \frac{1}{\sqrt{Nc_s}} \times \text{ (sum of } Nc_s \text{ whole } x\text{'s) } (s = 1, 2, \ . \ . \ . \ n) \ . \ . \ (10)$$

where c_s is the proportion of the whole number of x's that enter into q_s. In other words, every l in equation (8) becomes in the special case of dice throws either zero or the same as every other l that is finite.

Now suppose that the x's in the several q's are chosen by picking out Nc_1 x's at random to form q_1; then putting these back and picking out Nc_2 x's to form q_2; and so on. Then it has been shown that this may be done so that the proportion of x's common to any group of q's and to no others (*i.e.*, occurring in each member of the group and in no other q's), would be equal to the probability that any particular x was common to that group and to no others; and that, in this case, the correlation, r_{st}, between q_s and q_t is $r_{st} = \sqrt{c_s c_t}$ so that $r_{as}/r_{at} = r_{bs}/r_{bt}$ for all values of $a, b, \ . \ . \ . \ s, t, \ . \ . \ .$ from 1 to n. In this case, therefore, there is a perfect hierarchy. It follows that the q's are compounded, according to equation (3) above, of a single general factor and specific factors only. It may be shown that the same tends to be true when the x's in q_s are selected as in the example described on pp. 175 and 176 of the book under review. The resulting approximation to hierarchical order is due to the very special form of equation (10) as compared with the general equation (8) and to the manner in which the x's in equation (10) are selected. And yet the

authors conclude that since "Dr. Hart and Professor Spearman would . . . claim the hierarchy as being a sample of a very perfect one," which it is, "this example goes far towards shaking confidence in their criterion." We cannot agree. This example does not affect the consequences that follow when Hart and Spearman's conditions are satisfied—that is when the correlation of every pair of columns in a square table of correlation coefficients is ± 1; still less does it throw any doubt on the truth of the fundamental proposition that when there is a perfect hierarchy there is a single general factor and specific factors only.

But the formula employed by Hart and Spearman in order to correct "observed" coefficients of correlation may be faulty, as our authors argue. In that case further experimental tests of dissimilar mental qualities in large numbers of individuals will still be needed in order to settle the question whether the correlations of these qualities form hierarchies (as was very approximately the case with both the series of tests which Cyril Burt made on school boys in Oxford) and so demonstrate that there is present a single general factor in every quality tested.

We emphasize the need for testing dissimilar qualities in these experiments, because group factors will always be present in every group of similar qualities. In intellectual qualities, for example, it is convenient to recognize a group factor, "cleverness," and in moral qualities another group factor having some close relation to "purpose." These group factors are, by definition, independent of each other and of the single general factor. They may be important. But the book under review surely errs when it says (p. 173) that "Mr. Maxwell Garnett . . . concludes that in addition to the single general factor of Professor Spearman, there are two large group factors which are practically general." It is true that Webb, who first noticed one of them, called it a "second general factor." But would he or anyone else expect either of these group factors to appear in qualities—in sensory discrimination, for example—which are outside their own special groups, but which may still depend upon the single general factor?

It should be noticed that Thomson's sampling theory of ability (equation (10)), and Spearman's theory of general ability (equation (3)), are by no means mutually exclusive. If further experimental investigations confirm the existing evidence that the correlations of sufficiently dissimilar mental tests tend to form hierarchies, we shall be able to say with equal truth that the several qualities may be regarded as compounded (according to equation (3)) of a single general factor—general ability—and specific factors only; or that each quality is composed (according to the all-or-none law in equation (10)) of a sample of a very large number of distinct elements. Time will show which of these two conceptions will prove to be the more fruitful.

MAXWELL GARNETT.

Syphilis und Nervensystem. Von Dr. MAX NONNE. Vierte, neu-durchgesehene und vermehrte Auflage, mit 169 Abbildungen im Text. Pp. 1,019. Berlin: Karger, 1921.

This well-known work, consisting originally of a series of lectures, has been thoroughly revised and brought up to date. It still remains the most comprehensive account of the effects of syphilitic infection of the nervous system, and we have not been able to discover any aspect of the subject that is not thoroughly considered in the light of recent research and clinical experience. It is well printed on remarkably good paper and should be in the possession of every neurologist.

Pathology of the Nervous System. By E. FARQUHAR BUZZARD, M.D., and J. GODWIN GREENFIELD, M.D. Pp. 334, with numerous illustrations. London: Constable, 1921.

Both the senior student and practitioner have long desired a short but authoritative account of those pathological changes which accompany common diseases of the nervous system. This book exactly fills such a want. It is clearly written, and the information given is accurate and in advance of that found in textbooks. The illustrations are admirable; they are excellently reproduced from original photographs of specimens prepared by the authors. Each chapter is followed by a short series of references to such literature bearing on the subject as is accessible in any medical library. This little book can be highly recommended to all who are anxious to acquire a sound knowledge of the pathological anatomy of the nervous system.

The Basis of Psychiatry (Psychobiological Medicine). By ALBERT C. BUCKLEY, M.D. Pp. 447, with 79 illustrations. Philadelphia and London: Lippincott, 1920.

The first part is devoted to a somewhat commonplace account of the functions of the central nervous system and the biological phenomena underlying heredity. This is followed by a classification of mental diseases according to their causes, manic-depressive insanity and paranoia excepted; for instance, under alcoholic psychoses nine separate varieties are described. In the chapter dealing with methods of examination we are not told how to make observations on the mental state of the patient, but there is a full account of the technique of the Wassermann reaction. Dementia præcox and paranoia, those touchstones of a textbook on psychiatry, are described purely from the aspect of the patient's external behaviour and no attempt is made to analyse the various psychical factors which go to make up his mental state.

The Clinical Examination of the Nervous System. By G. H. Monrad-Krohn, M.D.Christiania, M.R.C.P.London, Lecturer in Neurology at the Royal Frederick University, Christiania. With a foreword by T. Grainger Stewart, M.D., F.R.C.P., Physician to Out-Patients, National Hospital for the Paralysed and Epileptic. Pp. 135, with 12 diagrams. London: H. K. Lewis, 1921.

This little volume contains a useful description of the more important methods to be employed in the investigation of mental and nervous functions. The author's choice of tests is excellent and he has incorporated in this book the results of much recent neurological work. He rightly lays stress upon the necessity for linking up psychiatry with neurology, and insists on the importance of investigating the mental functions during the routine examination of the nervous system. The student will find this volume a most useful companion both during his work in the wards and subsequently in practice.

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EDITOR.

[JANUARY, 1922.]

BRAIN.

PART 4, VOL. 44.

DISTORTIONS OF THE VISUAL FIELDS IN CASES OF BRAIN TUMOUR.

(*Sixth Paper.*)

THE FIELD DEFECTS PRODUCED BY TEMPORAL LOBE LESIONS.[1]

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INTRODUCTION.

TEN years ago when this series of papers[2] was projected, accurate perimetry as we understand it to-day was rarely employed; and, indeed, as an instrument of precision the perimeter was rather an ornament and object of curiosity than an instrument of daily use in ophthalmological and neurological clinics.

To be sure, for the sake of record, the fields were occasionally plotted when the presence of a hemianopsia was more or less obvious,

[1] Presented before the American Neurological Association, Atlantic City, June 14, 1921.

[2] The preceding numbers have been as follows:—

First Paper (with George J. Heuer): "Statistical Studies." *Johns Hopkins Hosp. Bull.*, 1911, **22**, 190-5.

Second Paper (with George J. Heuer): "Dyschromatopsia in Relation to Stages of Choked Disc." *Journ. Amer. Med. Assoc.*, 1911, **57**, 200-8.

Third Paper (with Clifford B. Walker): "Binasal Hemianopsia." *Arch. Ophthal.*, 1912, **41**, 559-98.

Fourth Paper (with Clifford B. Walker): "Chiasmal Lesions with especial Reference to Bitemporal Hemianopsia." *Brain*, 1915, **37**, 524-42.

Unnumbered Paper (with Clifford B. Walker): "Studies of Optic Nerve Atrophy in Association with Chiasmal Lesions." *Arch. Ophthal.*, 1916, **45**, 407-37.

Fifth Paper (with Clifford B. Walker): "Chiasmal Lesions with especial Reference to Homonymous Hemianopsia with Hypophyseal Tumour." *Arch. Ophthal.*, 1918, **47**, 119-45.

or when vision was impaired by a large scotoma. But the cases were infrequent and few clinicians cared to perfect themselves in the use of the instrument which was time-consuming and at best gave information of only secondary diagnostic importance.

There were, perhaps, other reasons to account for its neglect. Lesions affecting the visual pathway, whether from tumour, trauma or vascular disease, were more or less hopeless from a therapeutic standpoint, and, what is more, in the presence of tumour such field defects as might be of localizing value were usually obscured by the contractions due to the secondary optic atrophy usually present before the diagnosis of tumour was ventured upon. Then, too, pituitary tumours in which successive perimetric records are especially important were regarded as rare, and usually passed unrecognized.

All this has greatly changed, more so, I fear, than is generally appreciated, if one may judge by the imperfect readings, taken with a single test object and recorded on small charts, such as are commonly sent to us when patients are referred to the clinic. Our own early records were of this sort, but when compared with readings such as those taken by Dr. Walker while attached to the clinic, the difference is as great as that between a child's sketch and an architect's drawing to scale. One gives an impression often misleading; the other the exact details. And many of the early errors into which we were trapped, such as our mistaken views on colour-interlacing, have thereby come to be explained and avoided [6]. Dr. Walker's chief contributions to the subject, as set down in his independent papers [7, 8], lie in the direction of accuracy as well as of simplification, and of late we have come to rely for clinical purposes on quantitative perimetry with test objects graded down to the smallest visible size. By so doing we may safely disregard the recording of the colour fields, provided those for form are sufficiently exact, for, as he has stated, "Colour defects are practically always represented by form defects when the visual angle is made small enough." Though few can hope to have the experience and acquire the facility in perimetry possessed by Dr. Walker, nevertheless the time necessary for the examination may be kept within reasonable bounds and trustworthy fields of vision secured after some practice by any intelligent observer copying his methods.[1]

[1] It may be said that the perimetric charts introduced by Dr. Walker are large sheets the same size as the hospital history sheets. They have necessarily been reduced to such an extent for purposes of this publication that many of the figures indicating the size of the discs, which have been employed in plotting the fields, are illegible in the reproductions accompanying the text.—Ed.

The Temporal Loop of the Optic Radiation.

Before describing the disposition of the geniculo-calcarine fibres in the lobe, which is the matter of primary importance, the story given in the first paper in this series, as an illustration of the value of accuracy in perimetry, may deserve re-telling.

There entered the Johns Hopkins Hospital in 1910 a man subject to convulsive seizures, with a gustatory aura typical of fits of uncinate gyrus origin. Some years before he had been shot in the left eye, and, as a radiogram showed, the ball had lodged in the petrous portion of the left temporal

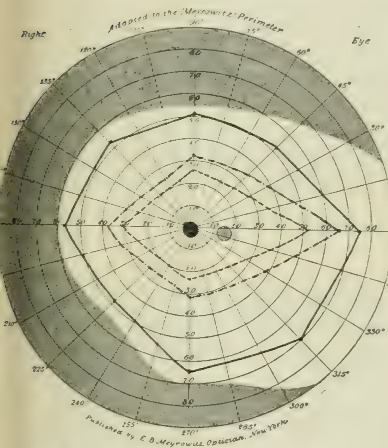


Fig. 1.—Chart of October 18, 1910, showing what were regarded as practically normal field relations. Note that eight points were relied upon for the form field and only four for the blue and red fields.

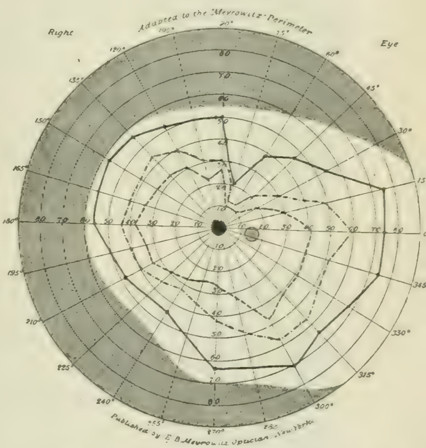


Fig. 2.—Corrected chart of October 19, 1910, for comparison with fig. 1, disclosing upper right temporal defect.

bone. Obviously the lower part of the left temporal lobe had been traversed. The field of vision of the remaining eye (fig. 1) had been plotted by Dr. S. J. Crowe, my then assistant, and it was regarded as normal. Indeed, no defect was anticipated, and the observation was made merely as the routine procedure of a painstaking house officer. The patient was shown one day on a ward visit which Dr. Adolf Meyer happened to attend, and he pointed out that the field periphery which had been plotted from tests at every thirty degrees might show a sector-shaped defect if taken with closer angulation, as only a few fibres of the pathway might have been damaged. This was done, with the result shown in fig. 2. Had the left eye not been destroyed, the defective sector would presumably have appeared as a homonymous one.

Three years before, in a paper which has received an undeserved lack of attention, Meyer [5] had described "the peculiar detour of the ventral portion of the geniculo-calcarine path which simulates the existence of an inferior longitudinal fasciculus." By the study of secondary degenerations following old vascular or traumatic lesions which had shown a fortunate limitation, either by exclusion or inclusion, to the visual pathway, he had observed that a portion of the optic radiation, on leaving the geniculate body, plunges far forward into the temporal lobe to sweep around the horn of the ventricle before it turns backward to end in the calcarine cortex. Though Meyer's findings were merely corroborative, in large part, of those by his predecessors—Flechsig, Henschen, Archambault [1], and others—he was able to demonstrate the course of isolated paths which show not only that the dorso-lateral and ventral bundles remain the same size throughout, but that they maintain in their course a definite position in relation to other bundles. Thus, the dorso-lateral bundle not only remains the most dorsal, but the most direct, all the way from the geniculate body to its end stations. The most ventral bundles, on the other hand, are the ones which make the longest detour around the temporal horn to end in the anterior part of the calcarine cortex.

A glass-model reconstruction of these fibres was made by Dr. Meyer, and it is from this that, at my request, he has permitted Max Brödel to make the superb diagrams (figs. 3, 4, and 5), which accompany this paper. They render superfluous any further written description.

Meyer's own observations were not supported or confirmed by perimetric tests, which were difficult or impossible in the clinical material of the type he had at his command. Nevertheless, he shrewdly surmised in connection with the few reported cases of quadrant hemianopsia in which small defects had been found post mortem that the dorsal bundles have an upper retinal distribution and hence correspond with field defects in the lower quadrants, whereas *per contra* involvement of the ventral bundles is responsible for defects of the upper field quadrants such as was shown in fig. 2. In other words, the disposition of the fibres in the geniculo-calcarine radiation is practically the same as that in the optic nerve itself. Whether or not this means, as I believe it does, separate representation on the cortex for different areas of the retina, need not now detain us, for our present concern lies with the course of the fibres through the temporal lobe and not with their calcarine end-stations.

From the series of cases to be reported, we shall see that tumours

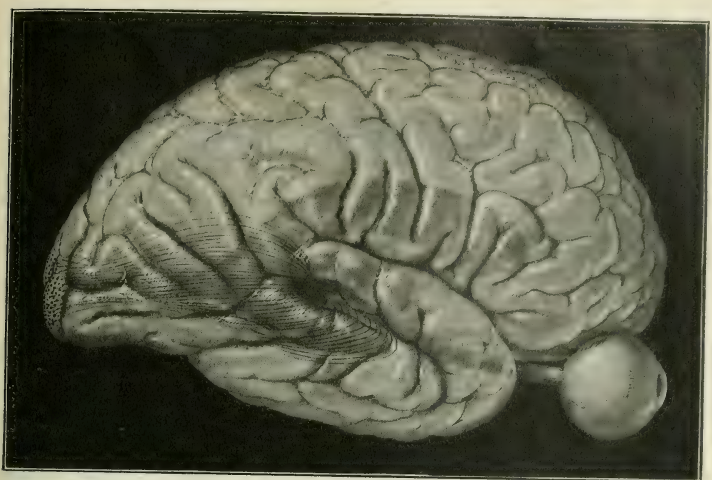


FIG. 3.—Showing the brain, as transparent, to indicate the lateral ventricle and the disposition of the fibres of the optic radiation.

FIG. 4.—Mesial aspect of the brain with disposition of the geniculo-calcarine pathway.

in the temporal lobe may produce homonymous field defects in the upper or lower quadrants which are of extreme value in localization. Lesions more posteriorly situated are in my experience less likely to

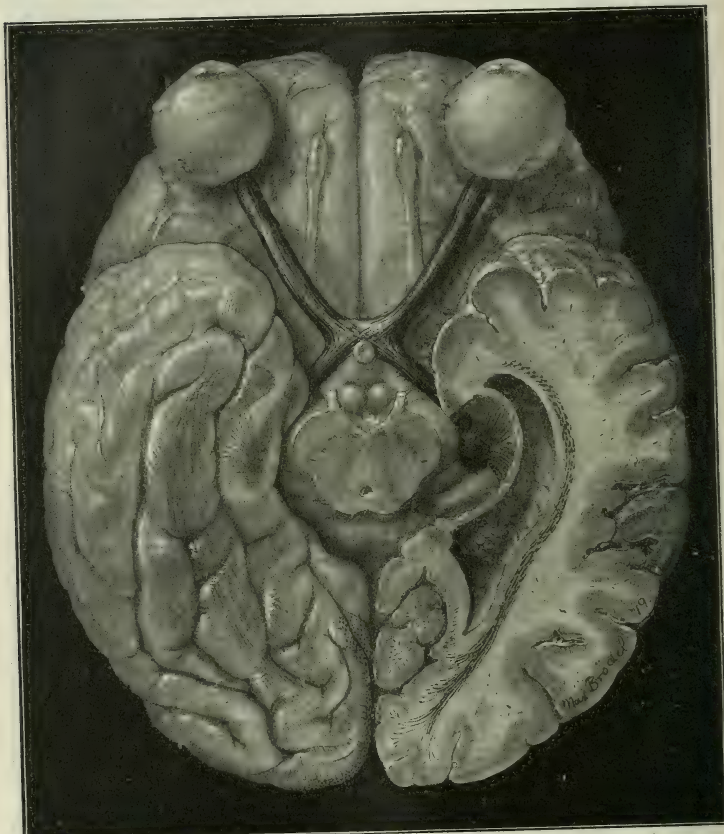


FIG. 5. —The radiation shown on the right in section and on the left in transparency.

produce these partial defects, or at least are less likely to be caught by the observer in a stage short of the usual midline hemianopsia.

SOURCE OF MATERIAL.

In the writer's series of 663 verified intracranial tumours, to April 1, 1921, there have been 197 tumours involving the hind-brain

(subtentorial), 190 pituitary tumours (including suprasellar lesions) and 276 tumours involving the cerebrum proper. Of this group of 276 cerebral tumours, in 59 the lesion has been situated largely within or has chiefly deformed the temporo-sphenoidal lobe. This by no means represents all the temporal lesions, but the doubtful cases have been excluded, since for our present purposes it is best to limit the discussion to those tumours which appear to have originated in the lobe itself.

Even so, that 59 out of 276, or over one-fifth of all verified cerebral tumours, should have primarily involved the temporal lobe may appear an unduly large number. For this, two explanations may be offered. One lies in the fact that, owing to the common practice of performing a right subtemporal decompression in tumours of doubtful situation, an unexpected temporo-sphenoidal lesion has not infrequently been disclosed.[1]

Another explanation is that tumour localization in this relatively silent area has of late years become more and more exact owing to the interpretation of field defects, which this communication, it is hoped, will serve to point out.

In 39 of these 59 temporal lobe cases perimetric charts sufficiently trustworthy for our present purposes were obtained and in many instances repeated observations over long periods have been made. In the remaining 20 cases the tests were untrustworthy or impossible for a variety of reasons, owing to practical blindness from secondary optic atrophy (six cases); owing to such a degree of stupor or mental confusion as to preclude dependable responses (eight cases); owing to other causes as aphasia, childhood, &c. (six cases). The fields of vision in the 39 dependable cases may be subdivided as follows into those showing :—

[1] It may need emphasizing that the cranial defect in a subtemporal decompression is made below the attachment of the muscle to the temporal ridge and consequently it is unusual to have more than temporal convolutions exposed. Occasionally one may barely expose the Sylvian vessels, but when there is a tumour of the lobe the Sylvian fissure is apt to be dislocated upward.

It is significant (1) that in the fifty-nine cases the lesion was found on the right side in thirty-two and on the left in twenty-seven instances; (2) that in only sixteen of the thirty-two right-sided cases was the lateralization helped out by perimetry. Some of the remaining sixteen had uncinate attacks and contralateral facial weakness assuring the localization, but the others were stumbled upon in the course of a routine decompression.

In a correspondingly large series of unverified tumours there is a long list of undoubted temporal lobe cases which have been excluded from this discussion. Many of these cases had uncinate seizures and field defects comparable to those in the verified cases, to the discussion of which this paper will be limited.

(1) *No defect* (*six cases*).—In these the fields were at all times normal. Three of them were slow-growing endotheliomata and one an angioma deforming the lobe from without, and therefore less likely to affect the pathway. That field defects may occur under corresponding conditions, however, some of the cases to be reported later on will make clear. A fifth case was a rare one of dermoid cyst in the tip of the lobe accompanied by uncinate seizures. From the situation of the lesion one would have expected a partial hemianopsia which repeated examinations failed to reveal. The sixth case was a glioma disclosed by a right decompression in which only a single preoperative test had been made.

(2) *Homonymous hemianopsia* (*eight cases*).—In these the condition had advanced to a median vertical separation of blind and seeing fields, indicating an interruption of the entire pathway, though the macular fibres were oftentimes spared. In none of these cases had fields been plotted sufficiently early to detect quadrantic defects, nor in any of them was a post-operative recession from the hemianopsia observed. The findings indicated that the cerebral lesion was on the right in five and on the left in three of these cases, but it did not otherwise indicate the position of the tumour in the hemisphere. It is presumable that in many or all of them the growth may have come to involve the geniculate body or even to have affected the optic tract itself by pressure. If reliance is to be placed on the sparing or otherwise of the macular bundle as an indication of the involvement of optic radiation *versus* optic tract it would appear that tract involvement may be expected as a late feature of all temporal lobe tumours. In four of these eight cases the macula was included in the field bisection at the time of the examination and in all likelihood in the course of time the macula came to be involved in all. Several examples of hemianopsia, both with macula spared as well as included, which occurred in the series, have been classified under the following heading for the reason that the patients showed at one time or another the partial defects (e.g., Case 7) with which we are chiefly concerned.

(3) *Partial hemianopsia* (*twenty-five cases*).—It is upon these defects, more or less quadrantic in character, that chief emphasis will be laid. At one time or another while under observation the patients all showed an homonymous defect, which fell short of a complete hemianopsia. As will be indicated by the case histories which have been selected for purposes of illustration, these partial defects have been observed either as: (1) a stationary condition; (2) a con-

dition advancing toward a hemianopsia ; or (3) a condition in process of recession after a successful operation.[1]

Case Reports.

At the outset one or two examples of some of the earlier cases in the series may be given. The fields were then taken with standard 5-mm. test objects, and many of the details of the more recent examinations are therefore wanting. These early observations, nevertheless, served to give us our first hints of the localizing value of these partial defects. This was particularly true of defects in the colour fields which were seen to precede those for form, a matter which is of less importance to-day, since practically the same information is secured by utilizing white discs of minute size. It was from some of these early cases, furthermore, that we first came to appreciate the fact that the field defect in the eye on the side of the lesion is apt to be in advance of the other.

Case 1.—*Gliomatous cyst of right temporal lobe producing a lower homonymous defect short of hemianopsia. Post-operative widening of fields to normal.* (J. H. H., Surg. No. 23551.)

January 30, 1909: Admission of S. G. W., a clerk, aged 26, with the complaint of headaches, fainting attacks and failing vision.

Chronology of symptoms: Always well until July, 1905, when he had a sudden loss of consciousness without warning or convulsive movements. One year later another similar attack, preceded by dizziness. Since then he has been " nervous " and somewhat less alert intellectually.

[1] On two earlier occasions, in 1912 with Dr. Heuer, and again in 1916 with Dr. Walker, preliminary steps were taken to assemble material for this paper, and to review the literature pertaining thereto. The first interruption came from my transfer to Boston, and the second was due to our entry into the war. To have been deprived of their collaboration is by no means made up for by the fact that the number of cases which can be drawn upon has greatly increased. My acknowledgments are due not only to them for a great deal of the preliminary work, but also to a succession of neuro-surgical assistants who will recognize many of their own charts and case reports in the text.

Our studies in 1912 were chiefly directed toward a determination of the value of Wernicke's hemiopic pupillary reaction, and also of Wilbrand's prism test in differentiating, in cases of pure hemianopsia, between an interruption of the pathway central or peripheral to the geniculate body. The tests, though sharply positive in some cases, were so obscure in others, that we subsequently have abandoned them, for at best the information they give is but confirmatory of what one may gain in other ways. Moreover, it is highly desirable to recognize and interpret field defects before a total hemianopsia has supervened and at that time these tests are not applicable. A homonymous hemianopsia, whether or not the macula is spared, which is accompanied by a primary optic atrophy, one may safely ascribe to a chiasmal lesion usually pituitary in origin: one which is accompanied by a choked disc probably involves the radiation somewhere in its course. This is not an invariable rule, but it is less likely to mislead than a dependence on the Wernicke and Wilbrand tests.

In August, 1908, onset of persistent headaches, chiefly nocturnal, increasing in severity. At about this time some disturbance of vision was first noticed, with occasional diplopia. Also, from time to time, flashes of light described as "luminous things" have been seen, but they are not lateralized. Also vague "dizzy spells" without loss of consciousness, but with no features suggesting an uncinate origin.

Physical examination: This was quite negative except for: (1) a choked disc of 3 D. right, with beginning atrophy and $\frac{20}{60}$ vision: 4 D. left, with $\frac{20}{30}$ vision; (2) an incomplete left homonymous hemianopsia (fig. 6). No hemiopic pupillary reaction (Wernicke).

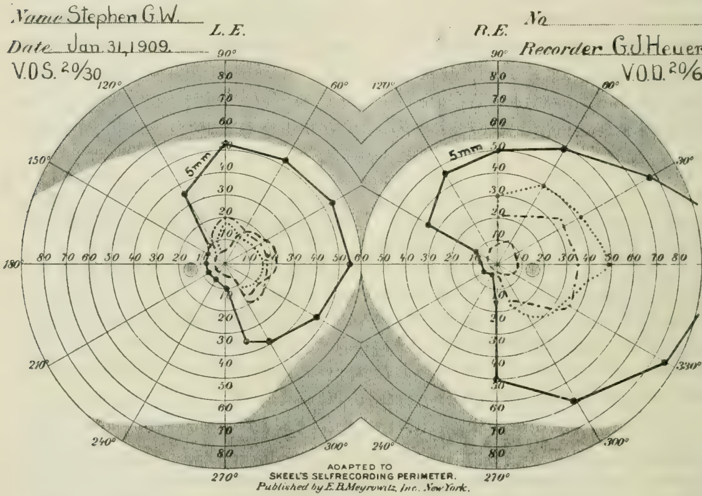


FIG. 6.—*Case* 1. Fields before operation which disclosed a gliomatous cyst of the right temporal lobe.

February 2, 1909: Operation.—Right subtemporal decompression. This disclosed a dry bulging temporal lobe which from surface appearances contained a subjacent lesion. A large gliomatous cyst pointing in the upper temporal convolution was widely opened and was drained for a week.

Post-operative: An immediate cessation of pressure symptoms. The choked discs subsided with gradual restoration of visual acuity to normal. Fields were taken every few days after February 10, on which date they showed normal outlines for form, though the colour fields were still slightly contracted (e.g., fig. 7). He was discharged March 4 with normal fields and vision of $\frac{20}{15}$ in each eye.

Subsequent note: Three years later (February 19, 1912) he returned for observation. He had been free from symptoms during the interval. The fields remained normal. After this the patient passed out of my hands. Two years later—nine years from the time of his primary loss of consciousness—he died with a recurrence of tumour symptoms.

Comment: When this patient was first seen, without the perimeter even a lateralizing diagnosis would have been impossible. It may be observed in the first charts (fig. 6) that there was a hemiachromatopsia on the right, the side of the lesion, the macula being spared. Doubtless the primary field defect began in the lower left quadrants, but it is improbable that the significance of this was sufficiently appreciated twelve years ago to justify us in anticipating the temporal lesion which was stumbled upon.

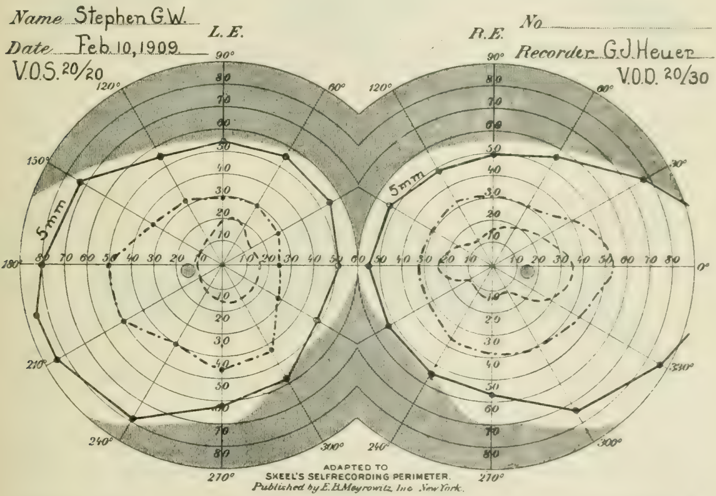


FIG. 7.—*Case* 1 (*contd.*). Fields eight days after operation, for comparison with fig. 6.

In the following case the conditions so far as the diagnosis was concerned were much the same as the above, though produced by a tumour of different type—a meningeal endothelioma. The case is one which interested us greatly at the time and was studied in detail, though the bare outline of the history must suffice for this report.

Case 2.—Large endothelioma involving right temporo-sphenoidal lobe producing a homonymous field defect unequal in the two eyes. Operation: Partial recession of field defect. (J. H. H., Surg. No. 30210.)

July 9, 1912: Admission of Louis C., a tailor, aged 33, complaining of headaches and failing vision.

Tumour history: For three years mild but more or less continuous headache referred to the right parietal region. For three months progressive failure of vision in the right eye. No other subjective disturbances. He had worked until the day of admission.

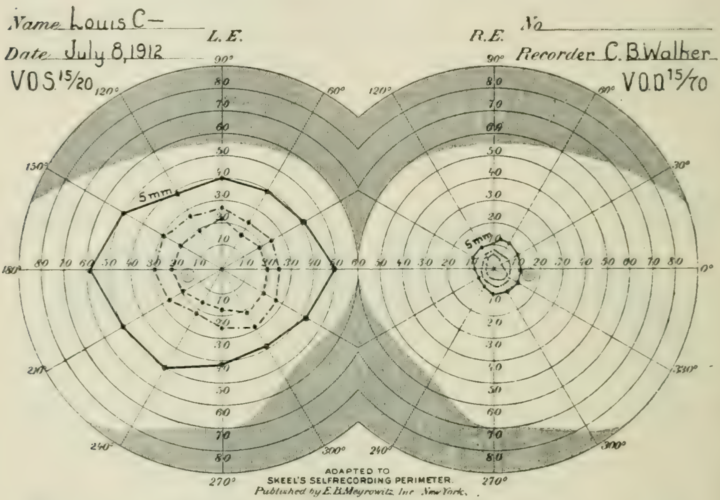


FIG. 8.—*Case* 2. Fields taken before operation which disclosed endothelioma of right temporal lobe.

Physical and neurological examination: Negative in all respects except for the condition of his eyes, which were slightly exophthalmic. There was a choked disc of 3 D. right and 2 D. left, with new tissue and some secondary atrophy, more marked right than left. The fields showed (fig. 8) concentric constriction, slight on the left but extreme on the right, where vision was practically tubular. V.O.S. $\frac{15}{20}$; V.O.D. $\frac{15}{70}$. The X-ray showed signs of general pressure but nothing of localizing value.

July 11, 1912: Operation 1: A right subtemporal decompression performed by my then assistant, W. E. Dandy, unexpectedly disclosed the edge of a tumour occupying the lower anterior part of the temporal lobe.

Post-operative: He made a good recovery with partial subsidence of his

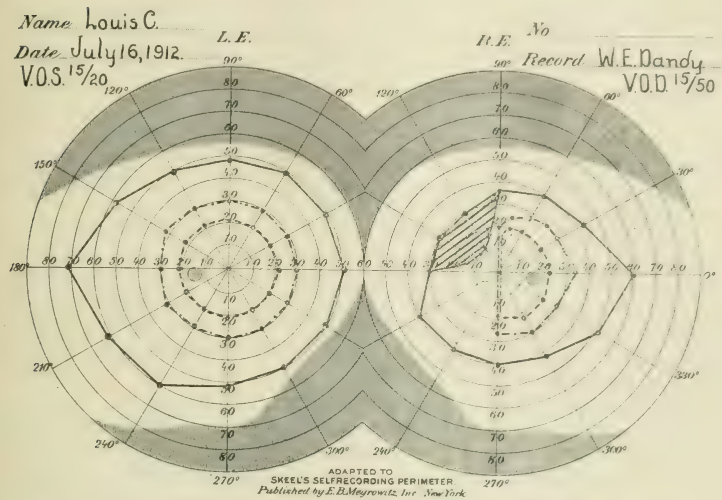


FIG. 9.—*Case* 2 (*contd.*). Fields five days after decompression which disclosed tumour. (Cf. fig. 8.)

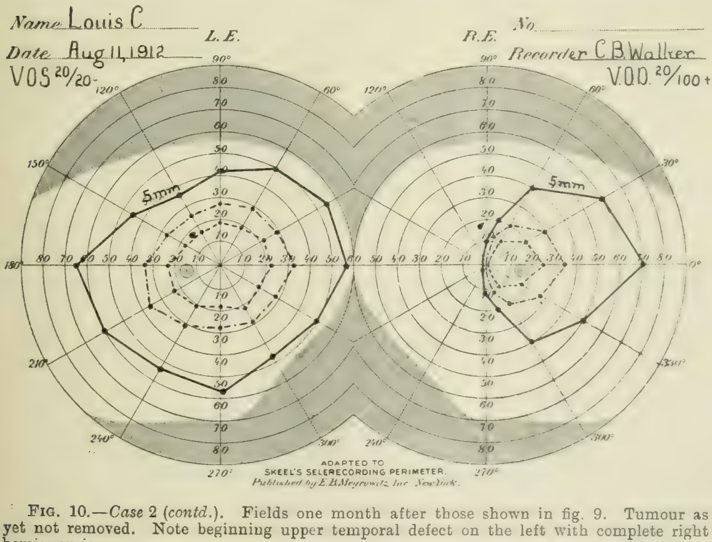


FIG. 10.—*Case* 2 (*contd.*). Fields one month after those shown in fig. 9. Tumour as yet not removed. Note beginning upper temporal defect on the left with complete right hemianopsia.

choked disc and lessening of his headaches. On July 16 the fields, which had widened, showed a nasal hemiachromatopsia with an upper form defect in the homolateral eye (fig. 9). He refused further operation and was discharged on this day to his home near by, but returned frequently for purposes of record.

By July 29 there was a fully established hemianopsia for both form and colour in the right eye, where further elevation of the disc had occurred. There was thought to be both a positive Wernicke and Wilbrand pupillary reaction, but negative by the method of Hess.

By August 11 the upper temporal field in the left eye had begun to be affected (fig. 10), indicating the beginning of a homonymous defect. His visual acuity on the right had become so much impaired that he expressed a desire to re-enter the hospital for further operation. The only additional symptoms observed and recorded at this time were a slight weakness of the left face and some subjective tinnitus referred to the left ear. The decompression area was tense and the discs measured about 2 D.

August 17 and August 26, 1912: Operation 2. Two-stage enucleation of a large endothelioma deforming the lower and anterior portion of lobe. He made a perfect surgical recovery from both procedures. On August 23, between the two sessions and before the tumour removal, the fields showed a still more marked upper temporal defect for the left eye. This was present also on August 29, three days after the extirpation, though by this time the fields had begun to widen out again (fig. 11). Fortunately, with subsidence of the choked discs normal vision returned for the left eye, though on the right at the time of his discharge the nasal hemianopsia persisted.

Subsequent note: Three years later, July 15, 1915, the fields were again taken at my request by Dr. Dandy (fig. 12). They show that in the interval there had been a slight filling out of the lower nasal quadrant in the affected eye. Four years later, October 27, 1919, visual fields kindly taken for me by Dr. Lloyd B. Whitham correspond exactly with those previously taken by Dr. Dandy. The process, therefore, may be regarded as stationary.

Comment: This case resembled the foregoing in that the examination revealed merely the general pressure symptoms of tumour and nothing whatever of localizing value if we except what the perimeter disclosed. Attention may be called to a number of interesting points.

(1) I am at a loss as to what interpretation to put on the unilateral tubular vision (cf. fig. 8), a condition commonly ascribed to hysteria. Four, or possibly five, other examples of the same condition, always on the side of the lesion, have been observed in the series of temporal lobe cases, and though we once felt that the finding might have some significance, I hesitate to lay any great stress upon the matter. (2) Of greater importance, though not observed till the tumour diagnosis had been made, was the gradual onset of the field defect in the homo-

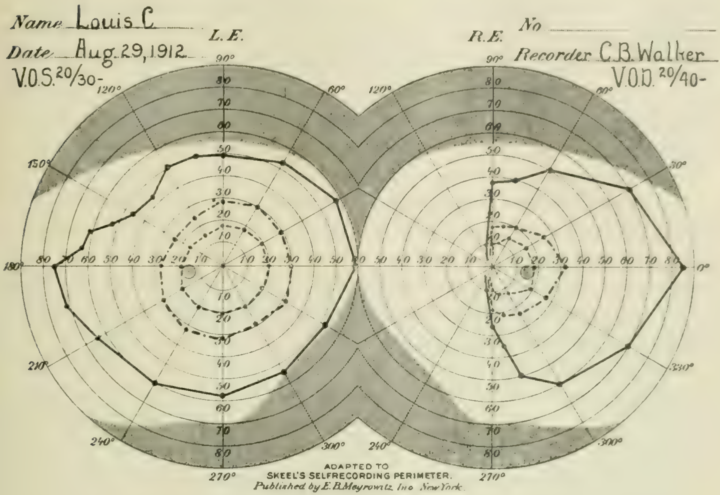


FIG. 11.—*Case* 2 (*contd.*). Three days after tumour extirpation. Note widening of fields compared with fig. 10.

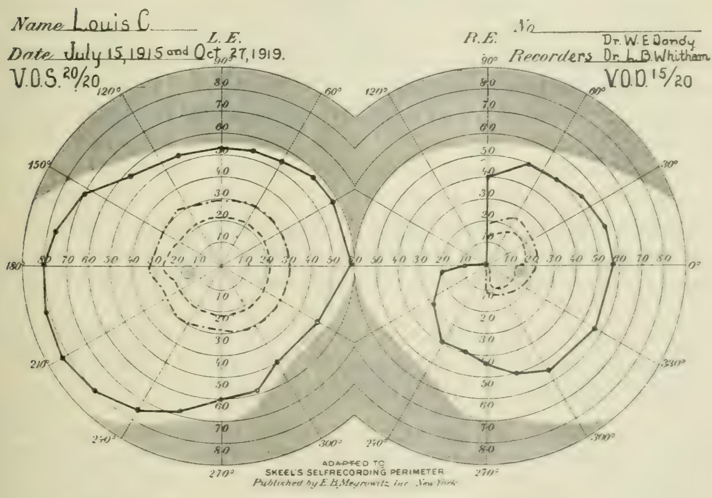


FIG. 12.—*Case* 2 (*contd.*). Present and stationary condition seven years after the tumour extirpation.

lateral eye, first showing as a hemiachromatopsia and advancing to a complete hemianopsia before there was any sign of a defect in the left eye. (3) There can be little doubt but that with graded discs these defects would have been much more apparent even in the left eye, and that a hemianopsia to form would have shown itself in correspondence with the hemiachromatopsia observed on July 16 (fig. 9), had a small test object been used below a millimetre size.

Confession must be made of one possibility of misinterpretation of these findings, particularly in consideration of the nature of the tumour and its extracerebral situation. For it is conceivable that the right optic nerve may have been pressed upon, a possibility made the more likely by the fact that the macula was impaired and there was a positive Wernicke reaction in the affected eye.

In answer to this possible criticism it may be said that pressure against the optic nerve by such a large tumour could easily have produced blindness but hardly a hemianopsia, and if the chiasma had been affected the field defects would have been more nearly equal in the two eyes. Moreover, as will be seen, a hemiopic pupillary reaction in our experience is not very dependable and the patient showed at the same time a positive reaction to the prism test which would indicate an involvement of the radiation alone. It possibly, however, is splitting hairs to discuss these differences, for, after all, the question of localization would be much the same, whichever interpretation is put upon causation of the field defects in such a case.

In the tumour cases subsequent to 1912, the more accurate perimetric methods developed by Dr. Walker have come to be utilized and have been applied to twenty-three of the thirty-nine temporal cases in which fields have been taken. In selecting the following examples from this later series, the attempt has been made to choose cases with lesions of different kinds in different situations in the lobe, as well as ones which illustrate the methods of advance as well as of recession of the field defects.

In the first of the cases which will be cited from this later series a gliomatous cyst was the offending agent. It was low-lying and, as will be seen, implicated the ventral fibres of the loop.

Case 3.—Gliomatous cyst of right temporal lobe producing left upper homonymous quadrantopsia. Operation. Recovery. (P. B. B. H., Surg. No. 582.)

November 14, 1913: Admission of Miss E. A. J., aged 43, with the complaint of headache and diplopia.

History: Subject to frontal headaches all her life. For six months there

have been severe attacks of pain referred to the suboccipital region. For four months considerable vertigo. Of late, some unsteadiness of gait which she ascribes to her diplopia. Also of late, paræsthesia of extremities and great despondency through a morbid fear of insanity.

On September 18, two months before her entrance, fields of vision taken by C. B. Walker disclosed a left upper quadrantal hemianopsia for form and colours (fig. 13). A month later, October 17, 1913, Dr. George S. Derby found the defect to remain practically unaltered.

Physical examination : Considerable exophthalmos, more right than left ; a slightly enlarged thyroid.

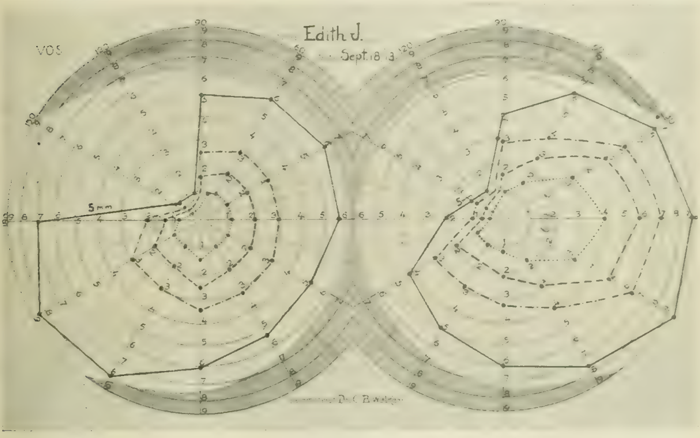


FIG. 13.—*Case* 3. Fields as found practically stationary during the two months before operation.

Positive neurological findings: A complete abducens paralysis. The eye-grounds showed slight hyperæmia with haziness of the nasal margins and overfilling of the veins ; no measurable swelling. The fields taken by Gilbert Horrax showed approximately the same defect observed on the previous tests. The condition, therefore, had been stationary for two months.

The examination otherwise was absolutely negative. There was no history of anything suggesting uncinate attacks. Nothing in the examination except her abducens palsy supported the suggestion of a cerebellar lesion given in the history of her complaints.

She remained under observation for a week, during which time she had no special discomforts. The perimetric findings favoured a right temporal lesion, but the diagnosis was uncertain and a choked disc questionable. She was discharged as a " brain tumour suspect " to report for observation.

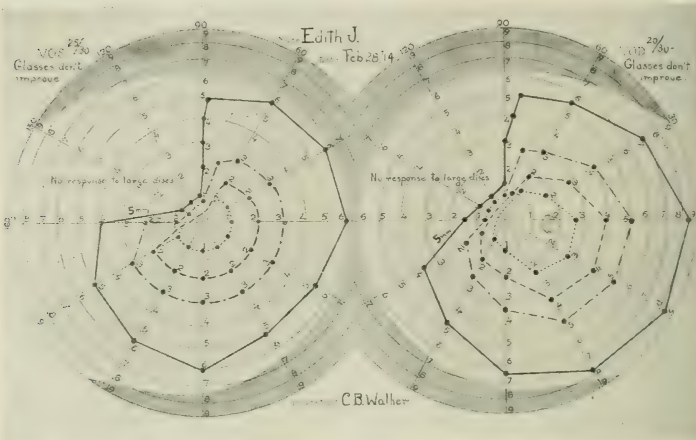


FIG. 14.—*Case* 3 (*contd.*). For comparison with fig. 13 five months later.

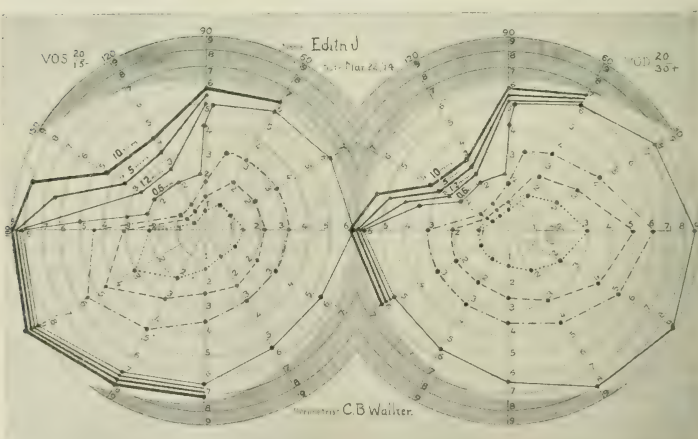


FIG. 15.—*Case* 3 (*contd.*). Fields sixteen days after operation disclosing gliomatous cyst in right temporal lobe.

During the next three months the condition remained unchanged except that her diplopia practically disappeared.

In February of 1914, she had several attacks of severe suboccipital pain with recurrence of the diplopia. This led to her re-entry. The fields were much as before, though the contraction was somewhat more marked (fig. 14). Haziness of the nerve head, however, was definitely more pronounced.

March 6, 1914: Operation. A right subtemporal decompression was performed. The temporal lobe was full, its convolutions flattened and an exploratory puncture through the second temporal convolution disclosed a gliomatous cyst containing about 30 c.c. of straw-coloured fluid.

She was discharged March 24, 1914, her mental condition having greatly improved on the subsidence of her headaches. The fields, as shown by Dr. Walker's charts (fig. 15), had begun to fill out to their normal outlines.

Following her discharge she remained free from symptoms for five years. The abducens palsy disappeared. Though from time to time the decompression area would become prominent and tight, this would soon pass off and there was no real headache. She had been active at social functions, and was engaged in the Y.M.C.A. corps during the war.

On the afternoon of April 16, 1919, while at the dressmaker's, she suddenly became nauseated and vomited. She was readmitted to the hospital three hours later. The decompression area was tense and her vomiting persisted. She began to have auditory hallucinations of people knocking at her door and of telephone conversations. There were visual hallucinations also—of seeing coloured lights and processions of queer figures marching on the ceiling. Her old diplopia again became marked.

Examination showed, as before, nothing but a low grade of papillœdema and a right abducens palsy. The fields of vision, though from inattention they were plotted with some difficulty, showed the same defects as on her first admission (fig. 13).

April 25, 1919: Operation 2. The temporal lobe was again exposed and the cyst was easily entered. It contained about 30 c.c. of straw-coloured fluid which set on standing. The walls were thoroughly fixed with Zenker's fluid until of a leathery consistency and the wound was closed. She made a perfect recovery, and the history records a recession of the field defects (the charts unfortunately have been lost). She was discharged May 22, 1919.

She reported in September, 1919, having been free from symptoms since her discharge. At this time the fields (fig. 16) still showed a slight notching. At the present time, two years from her last operation, and eight years since the onset of symptoms, she remains perfectly well, and her last perimetric examination, April 21, 1921, shows normal field peripheries (fig. 17).

Comment: This experience might be multiplied many times. The defect had never advanced to a hemianopsia but remained quadrantal, and on the side of the lesion, as is often the case, was a little in advance of the other. It involved chiefly the upper field quadrants, and hence indicated pressure on the ventral fibres of the pathway.

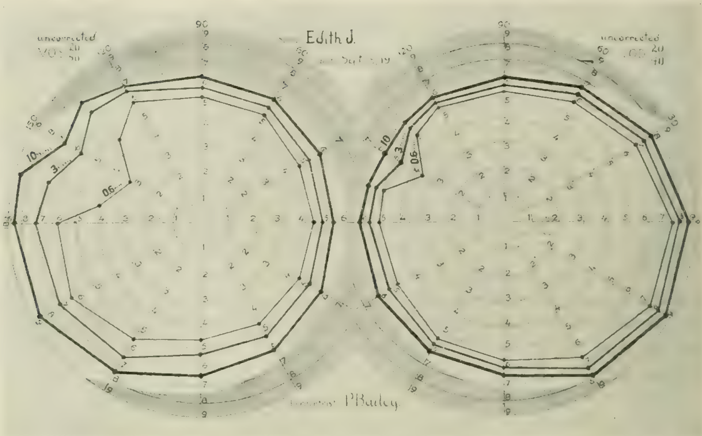


FIG. 16.—*Case* 3 (*contd.*). Showing persistence of slight homonymous defect five months after second operation.

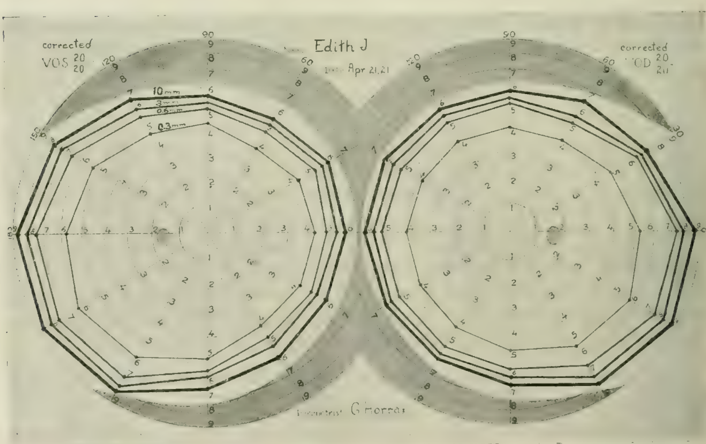


FIG. 17.—*Case* 3 (*contd.*). Normal fields two years after second operation.

Another example of this same type may be given, also a glioma, though one with a less favourable ultimate outcome. The patient was admitted at a time when Dr. Walker was making very detailed studies, as shown by the elaborateness of the first chart (fig. 18), which gives not only the form peripheries, but those for colours as well. It will be seen that there was a homonymous hemianopsia for colour as well as for form when the minute test objects were used. The condition, therefore, was somewhat more advanced than in Case 3, though the lesion was of the same type and in the same situation. A localizing diagnosis would not have been possible without the fields. They show still better than those of the foregoing case that the quadrantal defect on the side of the lesion, as shown by the larger discs, is somewhat in advance of that on the opposite side.

Case 4.—Homonymous hemianopsia produced by a gliomatous cyst of right temporal lobe. Operation followed by improvement. (P. B. B. H., Surg. No. 4550.)

April 8, 1916: Admission of Robert S., aged 36, a machinist, referred from the Boston Psychopathic Hospital, with the complaint of headaches and failing vision.

Clinical story: Four years before, he had been struck by a heavy piece of metal on the right forehead and rendered unconscious. There were no subsequent symptoms. For the past year he has not felt well, and has been drinking heavily. Six months before entrance (October, 1915) he had a general convulsion without warning. Since then there have been two similar attacks (November, 1915, and January, 1916). Since December he has had spells of suboccipital headache accompanied by nausea and vomiting. These have increased in intensity and there is now continued discomfort. For two months past there has been progressive blurring of vision. He has had numbness of arms and legs, some unsteadiness of gait, and also tinnitus in the left ear for the past year. He has had a few attacks suggesting *petit mal* in which he feels dazed and experiences a sensation of numbness and pricking in the right arm. Otherwise there has been nothing suggestive of uncinate attacks. He has been treated all this time for stomach trouble.

Physical examination. This disclosed nothing except the following few positive neurological findings. Bilateral choked disc of 5 D. with abundant hæmorrhages. Definite nystagmoid jerks on looking to the left. Slight impairment of hearing, i.e., conduction to watch less good on left than right (6 in. to 24 in.). Positive Romberg with falling to the right.

Aside from the evidences of a marked increase in intracranial tension the findings therefore were few. There was nothing to justify a localizing diagnosis, though it is evident from the history that the first examiner wavered between the suggestive cerebellar signs and the history of possible involvement of the right arm in the *petit mal* attacks. These possibilities were promptly set aside when the fields of vision came to be taken. They showed

a left homonymous defect which therefore lateralized the lesion in the right hemisphere beyond any diagnostic doubt (fig. 18).

April 19, 1916: Operation. Right subtemporal decompression. This disclosed an exceedingly tense temporal lobe. An exploratory trocar was inserted in the second temporal convolution toward the ventricle and at a depth of 3 cm. tapped a large gliomatous cyst containing over 30 c.c. of fluid. The tension was completely relieved. The cyst was widely opened by an incision through the second temporal convolution but its walls were not treated. A fragment of the wall removed for histological study showed a diffuse glioma.

He made a perfect recovery (figs. 19-20), and was discharged May 5, practically without symptoms, though the choked disc had not as yet com-

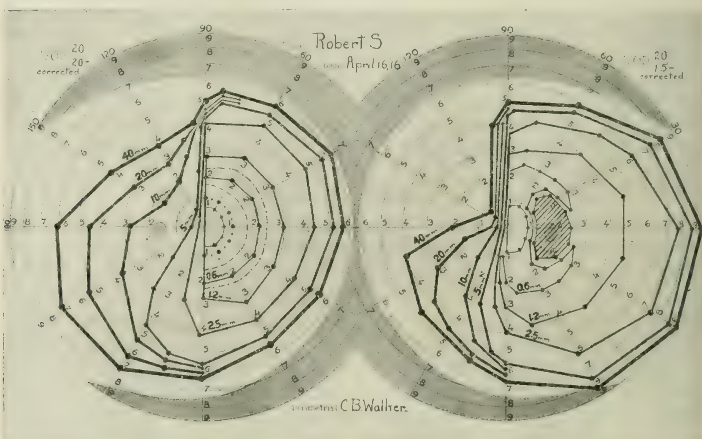


Fig. 18.—*Case* 4. Field taken before operation disclosing a right temporal gliomatous cyst. Note complete hemianopsia to discs below 1·2 mm. size. Also greater advance in defect to larger discs for right than left eye.

pletely subsided. The fields, except those for colour, had by this time partly regained their normal outlines (fig. 21).

December 22, 1916: Reported in person, having been working at his trade since shortly after his discharge. Condition excellent. No symptoms. Decompression soft. Fundi normal. Fields are normal except for a slight defect in the left temporal region to the smallest visible disc (fig. 22).[1]

[1] This patient re-entered the hospital October 22, 1918. Unfortunately no fields were taken at the time as the staff was mostly in army service. He had been quite well until the month previous and had worked regularly until a few days before. His decompression had become tense. The cyst was tapped on several occasions, but immediately refilled. An exploratory operation was performed which revealed an extensive gliomatous growth. He was discharged and his death was reported a few months later.

FIGS. 19 and 20.—*Case* 4 (*contd.*). Condition on discharge sixteen days after subtemporal decompression, showing slight protrusion at seat of defect.

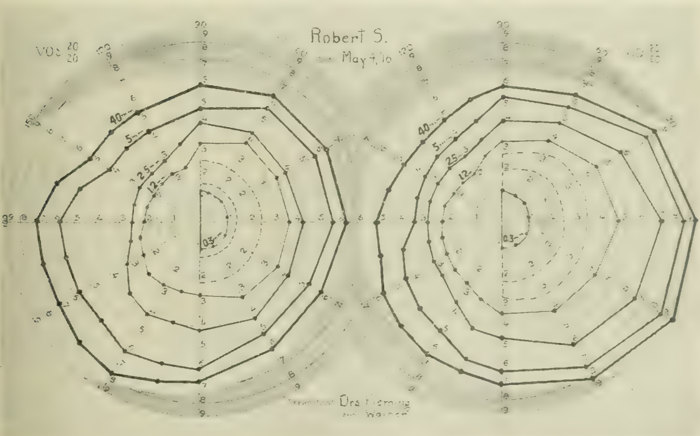


FIG. 21.—*Case* 4 (*contd.*). Fields on patient's discharge sixteen days after operation. Note persistence of homonymous hemianopsia to 0·3 mm. disc.

The following case, owing to the nature of the growth, was far less favourable than any of the preceding ones. The diagnosis of a temporal lesion was sufficiently clear owing to his full-blown uncinate seizures, though without the perimeter the lateralization of the tumour would have been difficult. At the time of his first admission, as will be seen, the fields were regarded as practically normal, though to a minute disc a notch in the right upper quadrant, the forerunner of a more extensive defect, was already apparent.

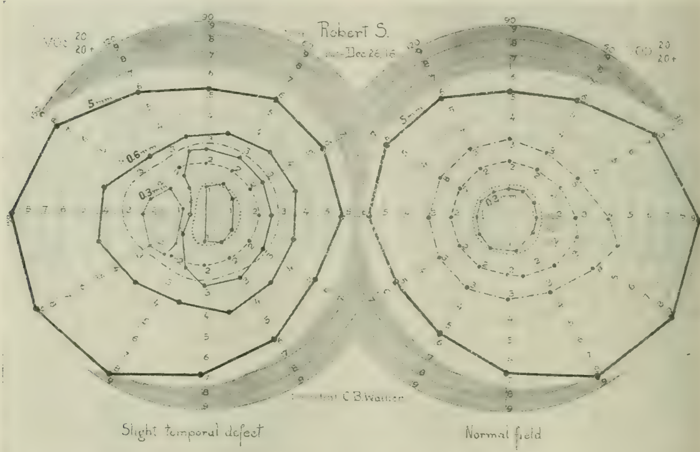


FIG. 22.—*Case* 4 (*contd.*). Fields six months after operation, for comparison with figs. 18 and 21.

Case 5.—*Solid glioma of left temporal lobe with uncinate seizures and an upper right quadrantopsia.* (P. B. B. H., Surg. No. 6273.)

February 16, 1917: Admission of Robert A. C., a physician, aged 36, referred by Dr. A. B. Kanavel, of Chicago, with the major complaint of *petit mal.*

History: Somewhat apprehensive about his health ever since his student days, he for fourteen years has regularly taken his temperature and in view of a customary afternoon rise to 99° has regarded himself as tuberculous. Frequent examinations by many physicians failed to corroborate this. The account of his *petit mal* attacks as taken in the records by Dr. S. J. Harvey is as follows:—

Uncinate attacks: "Eight and a half years ago began to have attacks of *petit mal.* The first one came on suddenly while taking a needle from a

patient's hand. He became dizzy, felt faint, had 'air hunger,' went to a window, opened it and soon revived. One month later there was a similar attack and soon they came with increasing frequency, often ten or twenty a day. The attacks are usually accompanied by what he describes as an exaggeration of his special sense impressions, i.e., flowers become extraordinarily beautiful, odours are intensified, his vision appears to be remarkably keen, so that objects seem to be increased in size. The olfactory hallucinations were never of an unpleasant nature.

"There has never been any loss of consciousness and the attacks have always been so fleeting that a bystander would hardly know he was having one. There is usually an aura 'as if something were to happen,' and during the attack he feigns to be meditating or cogitating over what may have just been said. He states that no two of the attacks are exactly alike but that they vary according to the time of day, his surroundings or the season of the year. For the past two or three years the attacks have been a little more severe; they would leave him momentarily dazed and disorientated; for instance, while talking to a friend he might be seized with an attack which would cause him to stop his conversation, and when he 'came to' would have to ask what he had been saying, or at other times would plead an important engagement to cover his confusion. He has occasionally had a sense of seeing objects to his right which were not there. For some years he has taken bromides in large doses, 70 to 80 gr. per diem."

Headaches: For fourteen months he has had occasional severe sub-occipital headaches but without vomiting. During these periods there has been some retraction and rigidity of the neck.

Vision: Has never noticed any diminution of vision, but he had glasses fitted when his headaches came on and found he had been seeing double without being aware of it. For a month he has had considerable bilateral tinnitus.

Physical examination: A robust individual over six feet in height and weighing 230 lb. The findings were absolutely negative except for a low grade of choked disc with swelling of 1 D., a fairly well marked lateral nystagmus, a slight weakness of the right face on expressional movements (discounted by the patient as a personal characteristic) and a slight weakness of the left abducens.

The fields of vision taken at this time with only two test objects (fig. 23) showed to a small 0·3 mm. disc a notch in the right upper quadrant of the left eye, whereas the peripheries to a 5 mm. disc were normal. No special significance was attached to these fields.

It is evident from this early history that the case despite the uncinate seizures was regarded, on the basis of the suboccipital headache and the nystagmus, as a cerebellar tumour suspect. He returned to his home and re-entered the hospital a month later. At this time his choked disc had become more pronounced with elevation of 3 D.

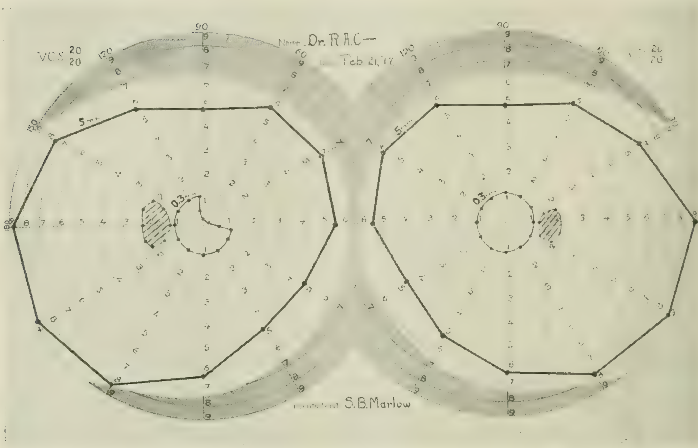


FIG. 23.—*Case* 5. Fields three weeks before operation. Note upper nasal notch in left to 0·3 mm. disc. Not regarded at time as of particular significance.

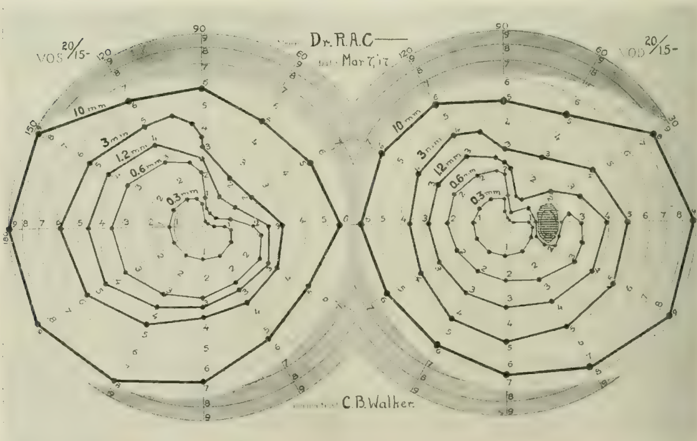


FIG. 24.—*Case* 5 (*contd.*). Fields showing homonymous involvement of which those in fig. 23 gave promise. Note notch at this session to the 0·3 mm. disc in each eye.

and the fields taken by Dr. Walker (fig. 24) showed an advancing defect, the right field now being involved. There remained no doubt as to the situation of the lesion in the left temporal lobe.

March 10, 1917: Operation. Left subtemporal decompression disclosing a tense bulging temporal lobe with flattened convolutions. Exploratory puncture secured about 2 c.c. of yellowish fluid. Diagnosis: glioma.

Subsequent notes: He made a good recovery. The choked disc subsided. He was discharged March 24. During my absence in the Army he reported by letter on October 10 as being in good health despite his continued *petit mal* attacks. He is reported to have died January 8, 1918. It may be assumed that before the end he had a complete homonymous hemianopsia.

The following case illustrates the terminal condition of complete hemianopsia which in the preceding would probably have been observed had a perimetric test been made toward the end. The patient had visual hallucinations referred to the defective fields (left) and a few gustatory impressions suggesting uncinate stimulation, though there were no "dreamy states." The lesion, therefore, even without the field defects, might possibly have been localized in one or the other temporal lobe, but the characteristic fields made its lateralization possible.

Case 6.—*A rapidly growing cystic glioma of lower right temporal lobe producing upper quadrantopsia unrelieved by operation and advancing to hemianopsia.* (P. B. B. H., Surg. No. 4002.)

December 14, 1915: Admission of Jacob B., aged 11, a right-handed boy, with the complaint of headaches and vomiting.

Clinical history (notes by Dr. Horrax): "Onset a year before admission, with frontal headaches and pain in the eyes, chiefly the right. The headaches were usually associated with nausea, vomiting, and often with dizziness. These symptoms have increased until of late headache has been constant. For a month he has had buzzing in the right ear, and he recently was operated upon in a local hospital for adenoids. Some occasional blurring of vision has also been observed during the past month, but his acuity remains unimpaired. For the past week there has been some double vision, and on three occasions he had had definite visual hallucinations, always to the left. Once he had the impression of seeing a boy on the wall. Another time a man dressed in white was seen sitting by the fire bending over to tie his shoe, and this impression remained a long time. One night, after his admission, he thought he saw some children sitting around a desk on the wall, but they were gone when the nurse turned on the light.

"On one occasion, just before entrance, he thought that he had smelled and tasted peaches when none were around, and after his hospital admission he insisted that he smelled and tasted roasted peanuts. These two occasions

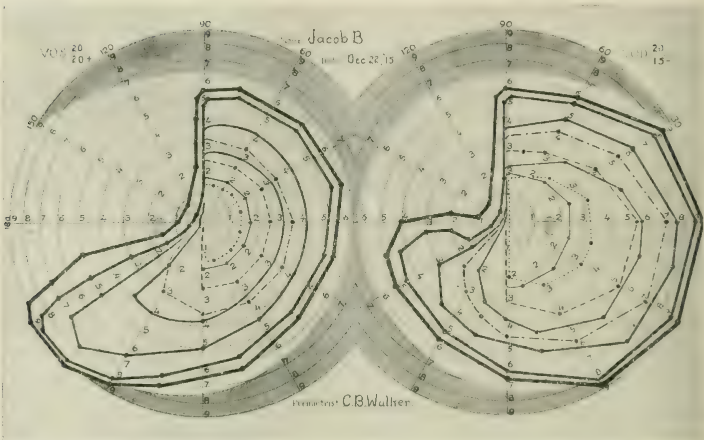
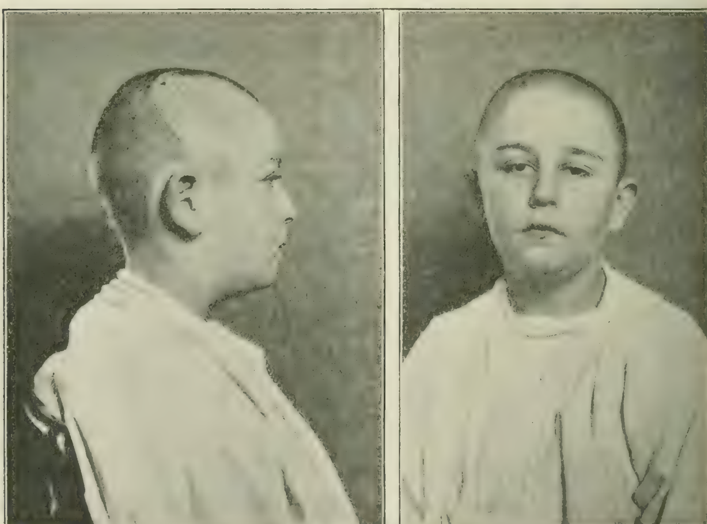


FIG. 25.—*Case* 6. Fields shortly before operation disclosing a right temporal glioma.



FIGS. 26 and 27.—*Case* 6 (*contd.*). Two weeks after operation showing situation of bone flap and slight temporal protrusion through the subtemporal defect.

were not associated with anything resembling a 'dreamy state.' He had never had any known losses of consciousness."

Physical examination: A healthy, intelligent, and co-operative boy, with no positive neurological findings except for a choked disc of 3 D., signs of intracranial pressure shown by the X-ray, and a left upper homonymous quadrantopsia (fig. 25).

December 31, 1915: Operation. The right hemisphere was exposed by an osteoplastic flap carried well down in the temporal region. Here the bone was found greatly thinned. The dura was opened over the temporal lobe and reflected upward. The convolutions were not greatly flattened, nor was the Sylvian fissure pushed upward. Palpation of the lower part of the temporal lobe gave the impression of a subjacent cyst. An exploratory puncture in the second temporal gyrus at a depth of 4 cm. reached a small cyst, giving a few cubic centimetres of typical yellow fluid. A subtemporal bone defect was made and the flap replaced.

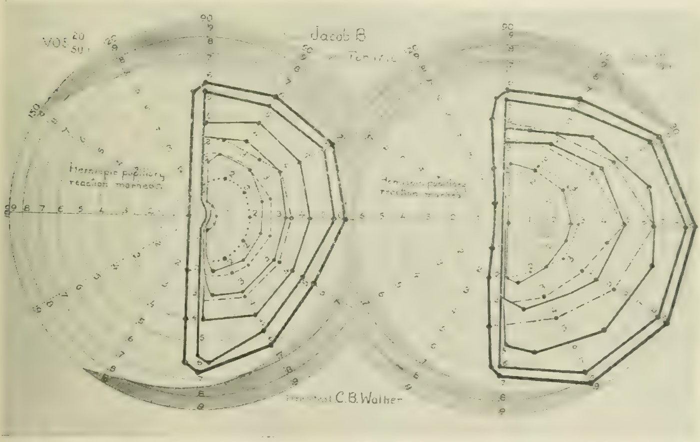


FIG. 28.—*Case* 6 (*contd.*). Fields two months after operation, for comparison with fig. 25, showing ultimate complete homonymous hemianopsia with macular inclusion.

The boy made a perfect recovery (figs. 26, 27), with relief of all subjective symptoms, except that once during his convalescence he had another vivid hallucination—of seeing "a lady wearing a hat." His choked discs subsided; but the fields of vision remained unaltered.

He was discharged January 22, 1916, much improved, but his period of relief was short. The decompression gradually became tense, and he began to have some left-sided numbness and weakness.

He re-entered the hospital February 16, 1916, owing to the increase of symptoms. His fields at this time (fig. 28) showed a complete hemianopsia. An operation was performed, and a large mass of gliomatous tissue containing small cysts was removed from the temporal lobe. There was again a temporary period of improvement, and he was discharged. The prognosis was hopeless, and he died a few months later at his home.

Doubtless, in a case of this kind, the geniculate body itself comes to be involved in course of time, and in all probability could a series of fields have been taken, there would have been a stage in which the macula was not completely bisected.

The patient whose history follows was first seen at such a stage, and there was considerable subsequent improvement, though in the end a fatal issue. In this case, too, visual hallucinations were a pronounced feature, as the story will relate.

Case 7.—*Cystic glioma of right temporo-sphenoidal lobe, with hallucinations of vision and a lower left quadrantopsia. Operation. Temporary improvement.* (P. B. B. H., Surg. No. 3043.)

June 9, 1915: Admission of Mrs. Helen L., aged 33, referred by Dr. D. B. Steuer, of Cleveland, Ohio, with the complaint of "headache and optic neuritis."

Clinical history: *Petit mal* attacks. In 1911, four years before admission, during the course of her first pregnancy, she began having attacks of *petit mal.* In these attacks her left hand was involved and would feel swollen. They were followed by twitching of the fingers and drawing up of the arm and face. She was invariably conscious during these attacks. After the birth of her baby these seizures, which had occurred many times a day, decreased in number and gradually disappeared completely.

Headache and diplopia: In 1912 she began having headaches referred to the right frontal region. They have become increasingly severe, culminating in a particularly bad attack three months ago, since when they are said to have been less troublesome. In 1912, also, she had for two weeks a period of diplopia. The eye-grounds were examined, and a choked disc of 4 D. was found. This condition continued throughout 1913, during which time she was under the care of a succession of "specialists."

Grand mal attacks: On four occasions she has had a general convulsion with loss of consciousness. The first occurred in June, 1913, the others eighteen, twelve and ten months ago respectively.

Anosmia and hallucinations of smell: Immediately after the birth of her child in 1912, she found that she had lost the sense of smell, and during the past year she has frequently had olfactory hallucinations, occasionally disagreeable ones. She describes them as "like something from another world; nothing that is in the environment—I can't describe it."

Left hemiparesis: First noted two years ago, as dragging of the leg and tendency to drop things from the hand. The symptoms became more definite three months ago in association with the series of severe headaches. The face then became weak and the arm and leg still more so. Some loss of sensation on the left has also been noted.

Failing vision: Choked disc first noted three years ago, but vision not greatly impaired until two months ago. She is conscious of loss of vision to the left.

Hallucinations of vision: Present for the past two or three weeks. For example, the following quotation is taken from the history: "She repeatedly told the nurse on awakening that a woman friend of hers, whom she named,

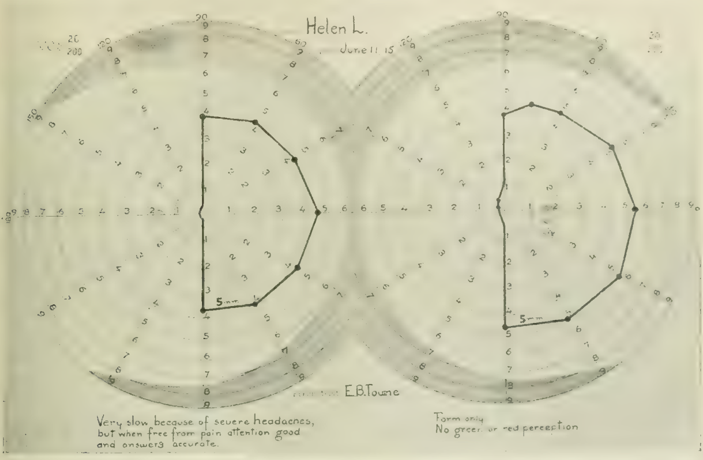


FIG. 29.—*Case* 7. Fields on admission shortly before operation.

was in the room, and she wanted her husband called to talk to this woman. The imaginary person stayed in the room all night, moving about, but did not talk. She was always to the patient's left. When the patient turned her head to follow her, the hallucination would also go to the left and disappear from the field of vision. To-day during the taking of the history she asked several times if the doctor had come back. Finally, she said, 'Well, who is it in the room?' On questioning it appeared that a man with a gold helmet on his head came into the room with the examiner and sat in a chair beside him. During the history-taking he also had a paper and took notes. He did not leave the room until the examiner left. His face was not recognized and she could not describe it clearly. Both examiner and this hallucination were to patient's left side."

Physical examination: A well-developed and nourished woman, showing the following positive neurological signs: a choked disc of 4 D., a total anosmia, a sharply-cut left homonymous hemianopsia (fig. 29). Hemi-hypæsthesia over entire left side of body with slight weakness of musculature. Deep reflexes increased to exaggeration on the left, with clonus and a positive Babinski.

June 14, 1915: Operation. Right osteoplastic flap, disclosing a tense dura. On exposing the hemisphere the temporal lobe protruded markedly and the Sylvian fissure was much dislocated upward. The convolutions were greatly flattened. A needle introduced into the posterior portion of the first temporal convolution struck a large gliomatous cyst containing nearly 100 c.c. of yellow fluid which clotted on standing. The tension completely subsided. This cyst was at such a depth that no effort was made to make an incision into it nor to treat its walls.

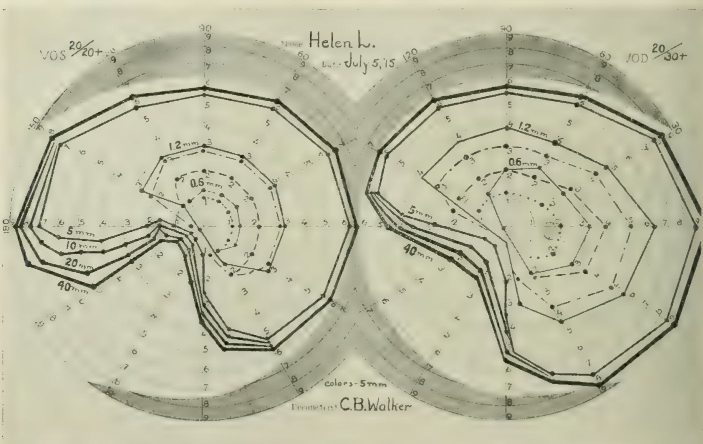
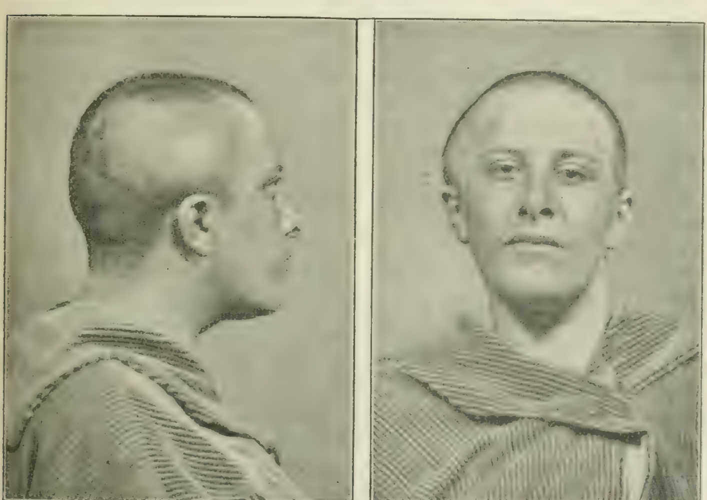


FIG. 30.—*Case* 7 (*contd.*). Fields three weeks after operation which disclosed a high temporal defect (for comparison with fig. 29).

Post-operative: For a few days she continued to have many bizarre visual hallucinations, always seen to the left. They were usually of people doing queer things, for example: "A woman riding horseback with a sheet of music propped in front of her and she used her hands as though playing the piano on the horse's neck." On one or two occasions also complained of disagreeable odours. These attacks subsided, however, and she made an excellent recovery with practical disappearance for the time being of all her previous symptoms. Normal reflexes, sensation and sense of smell were all regained. The choked

discs subsided with unexpectedly good vision in view of their original appearance. The fields taken on July 5, by Dr. Walker (fig. 30), showed a widening of the peripheries to the left with a persisting lower quadrantal defect which the high temporal situation of the lesion would have led one to expect.

She was discharged to her home July 9. Dr. Towne's final note is as follows : "Of late no convulsions, headache, hallucinations of smell, taste or vision. Left side now almost as strong as right, face still a trifle weak ; dynamometer right 24, left 18. Vision very much improved—cf. fields. Choked discs almost flat. No Babinski ; no ankle clonus. Deep reflex, still +, but less than before. Mental condition markedly improved ; still rather jocular but perfectly sane ; knows what serious condition she was in at entrance." (Figs. 31, 32.)



FIGS. 31 and 32.—*Case* 7 (*contd.*). Shortly before discharge, showing situation of osteoplastic flap and considerable protrusion through subtemporal defect.

Subsequent note : She passed into other hands and apparently no further fields of vision were taken. The cyst had to be tapped on several occasions. Her death was reported to have occurred a year after her discharge.

Even without the fields there should have been no real difficulty in making a diagnosis in this case, though the early attacks of *petit mal*, said to have begun in the hand, might have led one to anticipate a lesion which originated in the paracentral convolutions. Her homony-

mous hemianopsia, however, was enough to place it below the Sylvian fissure; and that the lower part of the pathway at least was not destroyed is apparent from the fields of July 5, which showed a return of vision in the upper areas.

The combination of visual, gustatory and olfactory hallucinations in a temporal lobe lesion is deserving of comment. The growth was of such a size that it must have occupied a large part of the lobe and, as was noted at the time of the operation, the Sylvian fissure was markedly dislocated, a condition not infrequently observed in large temporal lobe tumours.

The following case, one with a more favourable outcome, is another example of a cyst in practically the same situation as the foregoing, though at the time of admission the defect had not advanced to the stage of hemianopsia.

Case 8.—*Gliomatous cyst of right temporal lobe producing a lower homonymous quadrantal defect. Operation. Recovery.* (P. B. B. H., Surg. No. 11283.)

October 8, 1919: Admission of Dorothy D., aged 19, with the complaint of fainting spells and headaches.

Four years previously (1915) she had a severe fall on the ice, striking her head. To this she attributes her present illness, for soon after she began having headaches. These headaches grew worse, and one day, a year after the injury, while at school she fainted. Subsequently, fainting attacks with slight convulsive movements occurred every two to three weeks.

In 1916, at the Boston City Hospital, a right subtemporal decompression was performed by Dr. E. H. Nichols. Though her discomforts were completely relieved thereby until a few months ago, the fainting attacks continued. They are preceded by a feeling of numbness over the whole body, but there is no gustatory sense impression. The convulsive features of the attacks, hardly apparent at first, have of late become more pronounced. A large subtemporal protrusion has gradually formed (cf. figs. 36, 37).

Physical examination: An intelligent, alert, co-operative young woman, free from discomforts, and in excellent physical condition. While under observation she had a convulsion, beginning with a vague stare. For two or three minntes she could respond to questions and denied the presence of any olfactory or gustatory impressions. The left face, then the arm and leg, began to twitch, and a general convulsion followed.

Positive neurological findings: A bilateral choked disc with beginning secondary atrophy. Nerve head on the right elevated 1·5 D. On the left 3 D. Slight hypæsthesia over the left side of body without appreciable motor involvement, though the deep reflexes were possibly more brisk on the left than right. The fields of vision revealed a lower left homonymous defect (fig. 33).

October 10, 1919: Operation. An osteoplastic exploration was made to expose fully the right hemisphere. The temporal lobe was bulging, and its

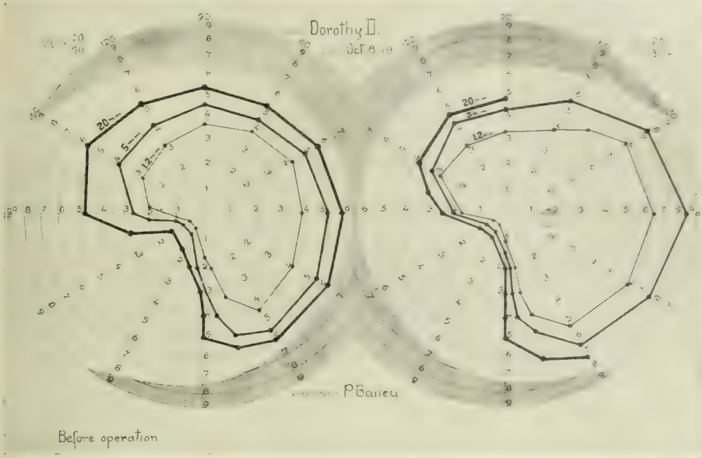


FIG. 33.—*Case* 8. Fields before operation which disclosed a high temporal lobe cyst.

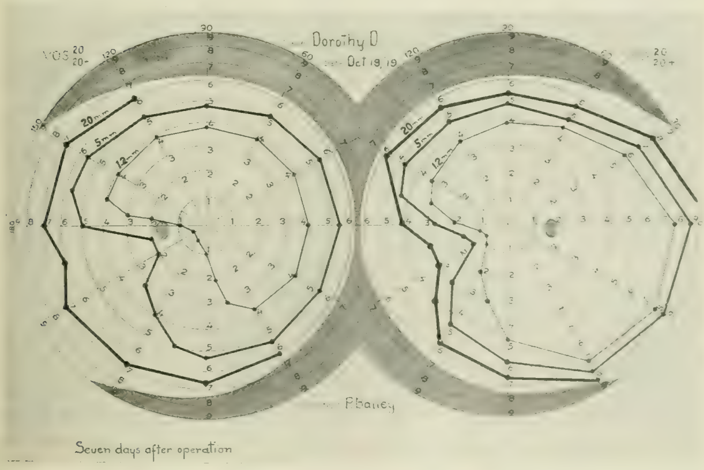


FIG. 34.—*Case* 8 (*contd.*). Fields nine days after operation, showing recession of defect.

convolutions flattened. Puncture of the first temporal convolution revealed a large cyst containing characteristic yellow clotting fluid. The cyst was widely opened disclosing a nodule of tumour projecting from its external wall directly under the old decompression. This nodule was completely excised. The cyst wall was treated as usual with a tissue fixative. The flap was replaced.

Post-operative note: She made a good recovery; had no more convulsions; the choked disc rapidly subsided, the hypæsthesia disappeared and she has remained perfectly well. The fields taken October 19, seven days after the operation, showed improvement, with widening of the peripheries to all discs and a definite filling in of the defect (fig. 34) to the larger ones. Ten days later (October 29), the day before her discharge, the field outlines were restored to the normal (fig. 35), where they have remained as shown by subsequent examinations (figs. 36, 37).

Subsequent note: She had one fainting attack Christmas, 1919. Since then no symptoms of her old trouble. Her most recent examination was made March 16, 1921, six years from the onset of symptoms. The decompression is now quite flat. Fields are normal. Fundus shows slight perivascular streaking: no swelling. She is steadily at work.

Comment: In this case, therefore, a cyst high in the temporal lobe and rather posterior exerted its pressure effect on the upper portions of the pathway, leaving the lower fibres unimpaired. The rapid restoration of the field peripheries show that it was a pressure effect only. There was nothing in her neurological examination to suggest a temporal lesion, except the perimetric findings, and in their absence as likely as not the temporal lobe might not have been exposed in the exploration. A puncture of the lobe at the first operation probably would have revealed the lesion.

The following history is one of an incurable glioma in which the field defects were seen to pass from a mild involvement through advancing stages to complete blindness.

Case 9.—Right temporal diffuse glioma with uncinate seizures. Successive operations. Advancing process. (P. B. B. H., Surg. No. 3774.)

October 28, 1915: Admission of Mrs. Sadie G., aged 28, referred by Dr. H. B. Eaton of the Boston Dispensary, complaining of *petit mal*, headaches and loss of vision.

Present illness: Dr. E. B. Towne's recital of her story may be quoted in full from the hospital record.

"*Petit mal* attacks: Began about a year ago. She says a peculiar feeling runs suddenly through the body, lasting only a few seconds. During this time her arms and legs feel numb and relaxed. She is told that she stares and that her face becomes flushed, and later pale. This happens possibly once a week. They are liable to come on while she is at the table. There is

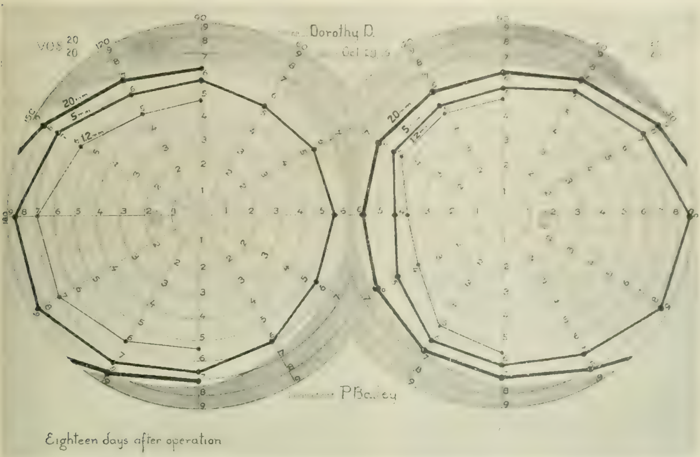


FIG. 35.—*Case* 8 (*contd.*). Showing normal peripheries on discharge nineteen days after operation, for comparison with figs. 33 and 34.

FIGS. 36 and 37.—*Case* 8 (*contd.*). *Circa* two weeks after operation, showing the position of the bone flap with soft protrusion at the seat of the old subtemporal defect, the scar of which is apparent and which directly overlay the tumour nodule.

never any loss of consciousness. She says she knows what is going on about her, but never speaks during the attacks.

"Hallucinations of smell accompany the attacks. The odour is described as like fresh paint and is so distinct she is surprised others do not detect it. It goes away immediately after the attack. She thinks that it has occurred possibly nine to ten times altogether, the last time about a week ago.

"Nausea: Began about eight months ago and comes two to three times a week. She has rarely vomited.

"Headache: She says it is really a pain on the top of her head rather than a headache. She calls it a 'pulling pain.' It has been in both frontal and temporal regions and in vertex. Of late it has tended to be more in the right temporal region than anywhere else. She is some days entirely free and again is miserable for two to three days. Headache is liable to be brought on by any sudden motion, as getting out of bed or sitting down suddenly?"

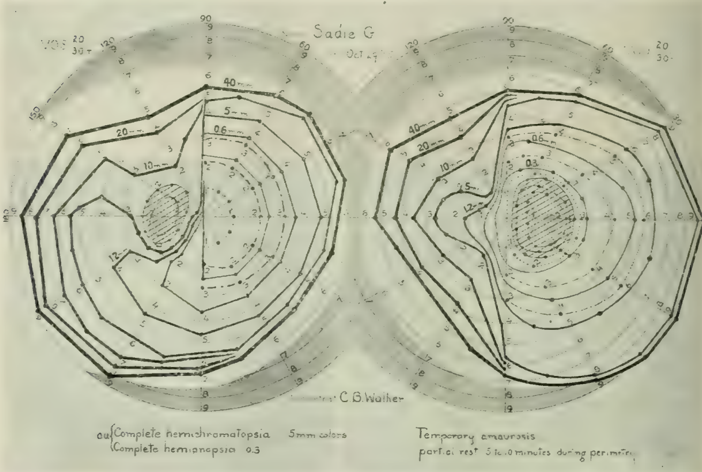


FIG. 38.—*Case* 9. Fields shortly before operation showing left homonymous hemianopsia complete in both eyes to a 0·3-mm. disc. Charts show unusually well gradations of discernible defect to larger discs.

Loss of vision: Four weeks ago she noticed that newspaper print was blurred and that she could read only with difficulty. Thinks that eyes were equally affected and that they have become gradually worse. At present cannot read print in telephone book. There have been attacks of temporary amblyopia quite frequently during the last two weeks.

Twitching of left face: During the past four weeks it has happened three

to four times. Corner of mouth draws up and eye closes. It is difficult to open eye again. It lasts a few moments. There is no twitching of arm and leg. No association with *petit mal*.

Hallucinations of vision: One week ago was walking across the room and thought that she saw a cat to her left. Says cat was very black and was hunched up as if angry. She turned around and looked about the room before she was convinced that the cat was not there. Two days later she again saw the same black cat on the left side. On two to three other occasions she has seen what she describes as black specks to her left, and she turns her head to look at them. There have been no other visual hallucinations.

FIGS. 39 and 40.—*Case* 9 (*contd.*). Patient on discharge, to show situation of osteoplastic exploration with subsequent protrusion through subtemporal defect.

Physical condition: A healthy, vigorous, intelligent, co-operative patient, sound in every respect. She was right-handed.

Neurological examination: This revealed absolutely no abnormalities except an advanced choked disc with hæmorrhage and exudates, and an elevation of 5 D. right and 6 D. left.

Her history was sufficient to suggest a temporal lobe lesion, but the side could not have been determined without the perimeter. An abnormality in the fields was first detected by Dr. Woodward, the interne, and they were subsequently plotted in great detail by C. B. Walker (fig 38). They showed an oncoming left homonymous hemianopsia with the process more advanced

in the right eye. To the 5-mm. discs there was a hemiachromatopsia which showed for form likewise when the 0·3-mm. test objects were used.

November 5, 1915: Operation. A low osteoplastic resection was made over the right hemisphere. The bone in the temporal region was thinned by pressure. A subtemporal defect was made to allow for subsequent decompression. The temporal lobe was full and bulging. Aspiration revealed no cyst. An incision was made through the first temporal convolution and carried down to a depth of about 3 cm., when it came upon a soft reddish tumour. A fragment removed for verification showed glioma.

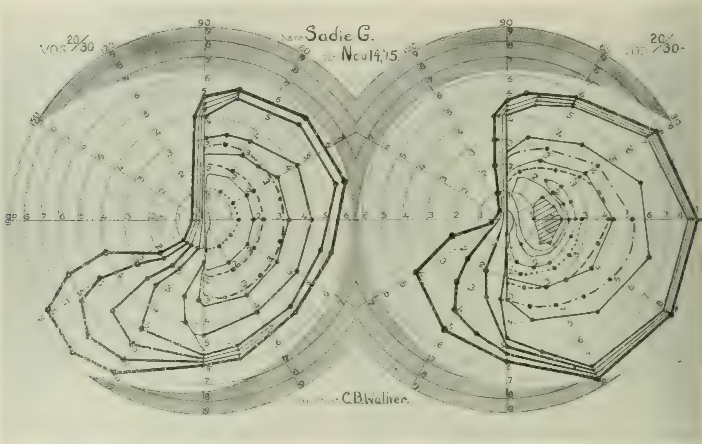


FIG. 41.—*Case* 9 (*contd.*). Fields three weeks after operation, for comparison with fig. 38, to show increasing field defects.

Post-operative: She did exceedingly well (figs. 39, 40). Headache and *petit mal* attacks ceased. The choked disc had subsided to 1 D. by November 26, the day of her discharge, The fields, however, showed a slight advance in the process (fig. 41).

In January, 1916, she reported subjectively free from symptoms. Dr. Walker's fields at this time (fig. 42) show a further slight advance in the process.

She remained perfectly well for nearly four years despite an increasing subtemporal protrusion. A note on January 24, 1917, states: "Patient reports in excellent condition. No symptoms. Says she never felt better in her life. There is a fairly large, rather soft subtemporal protrusion which she conceals perfectly with the arrangement of her hair (fig. 43). No further hallucinations or *petit mal* attacks. No fields taken."

March 10, 1919: She reported on my return from abroad in excellent general condition. No headache, hallucinations or *petit mal* attacks. The

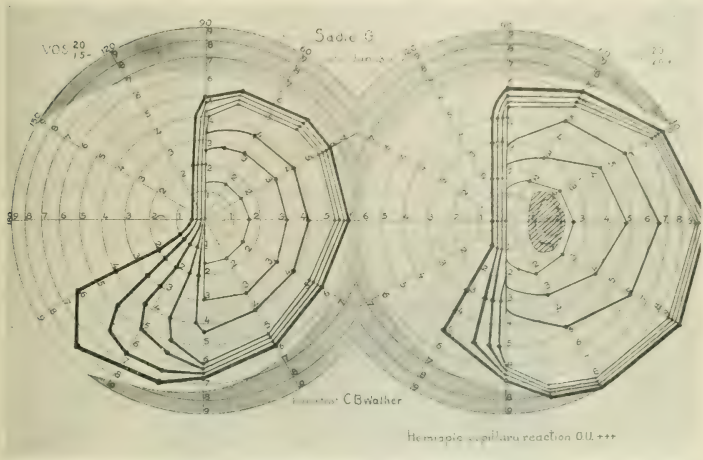


FIG. 42.—*Case* 9 (*contd.*). Field two months after operation to show further advance. (Cf. figs. 38 and 41.)

FIG. 43.—*Case* 9 (*contd.*). Patient fifteen months after operation, when she was subjectively free from symptoms.

protrusion, however, has been growing steadily larger and she has had occasional slight twitching of the left face. Vision in her left eye has been perfect until lately, and she is able to read without glasses. But from secondary atrophy the vision in the right is reduced to light perception. (No fields taken.) She was advised to re-enter the hospital for an exploration. It was hoped, in view of the slowness of the process, that the lesion had undergone cystic degeneration.

March 21, 1919: Re-entry. (Surg. No. 10165.) The process had advanced during the ten days. The protrusion had enlarged and the twitching of the left face had been replaced by a palsy (fig. 44). The vision was greatly reduced, shadows alone were seen in the right eye and in the left there was merely a hemiopic tubular vision (fig. 45).

FIG. 44.—*Case* 9 (*contd.*). Patient before last operation, showing huge protrusion with left facial palsy.

April 11, 1919: Secondary operation. A soft tumour mass the size of a fist practically extruded itself through the subtemporal defect on exposing the brain. As could be seen, this represented only the external part of the growth. Closure.

This procedure gave but little subjective relief and led to no improvement in vision. She died February 18, 1920, six years from the onset of her symptoms.

The patient's history on admission suggested a temporal lobe lesion in view of her visual and olfactory hallucinations. There was nothing

in the examination, however, to show whether the process was right or left, unless the slightly more advanced choking of the left disc could have been taken to favour this side. The visual fields on rough finger tests showed no hemianopsia, so that without the perimeter it would not have been possible to lateralize the process. The perimetric defects which were disclosed were typical of a low right temporal lesion affecting the ventral bundle of the radiation. As is usual the eye on the side of the lesion showed, throughout, the more advanced defect.

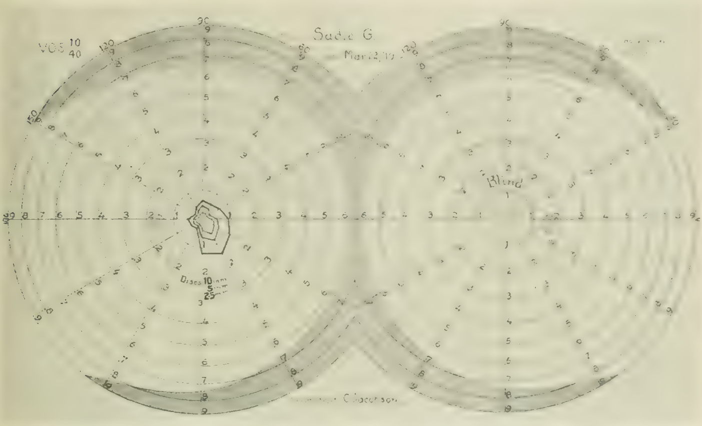


FIG 45.—*Case* 9 (*contd.*). Residual of vision on last admission four years following her first operation. For comparison with figs. 38, 41 and 42.

This peculiarity of an unequal progress in the defect on the two sides is still better shown in the next and last of the ten cases which have been selected for this report. The diagnosis was most obscure until the field defects began to appear.

Case 10.—*Brain tumour suspect. Decompression. Tumour localization made probable by early field distortions. Partial enucleation of meningeal endothelioma of left temporal fossa.* (P. B. B. H., Surg. No. 10671.)

June 19, 1919. Admission of Mrs. Ruth K., referred by Dr. E. O. Morrow, of Canton, Ohio, with the complaint of headache, vomiting and defective vision.

History: One year before entrance received a blow on the head and was temporarily stunned. Five months later began having suboccipital headaches, also pain over the left eye. Soon the eye began to protrude and turn inward.

Other complaints were transient numbness of left forehead, and pains referred to the left teeth. Morning nausea and vomiting frequent of late. Considerable dizziness and general weakness. Occasional subjective numbness in fingers of right hand.

Neurological examination : The only positive findings were a bilateral choked disc ; slight exophthalmos of the left eye ; left abducens palsy ; left pupil larger than right : slight expressional weakness of right face. A presumptive diagnosis of tumour involving the temporal lobe was made, possibly an endothelioma of the Gasserian envelopes. This was further supported by the disclosure, on taking her fields, of a notch in the upper nasal quadrant in the left eye (fig. 46).

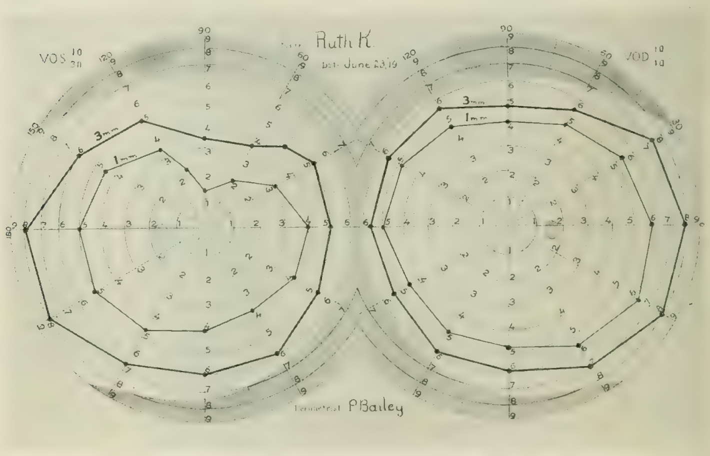


FIG. 46.—*Case* 10. Fields on admission of patient who was a left temporal lobe tumour suspect.

June 25, 1919 : Operation. Left osteoplastic flap. Combined exploration and decompression. The brain was found under practically normal tension. The ventricle was tapped to permit of a good view under the temporal lobe in the direction of the fifth nerve. Negative findings. The lobe seemed a little full but an exploratory needle introduced in two places revealed no lesion. Closure.

The symptoms remained unaltered. Occasional periods of numbness in the fingers of the right hand continued, accompanied sometimes by slight paraphasia. On July 7 she complained for the first time of sudden gustatory sensations—like peppermint. On this date the fields of vision showed a marked advance in the defect in the left eye and a homonymous notch in the right similar to that first seen in the left (fig. 47).

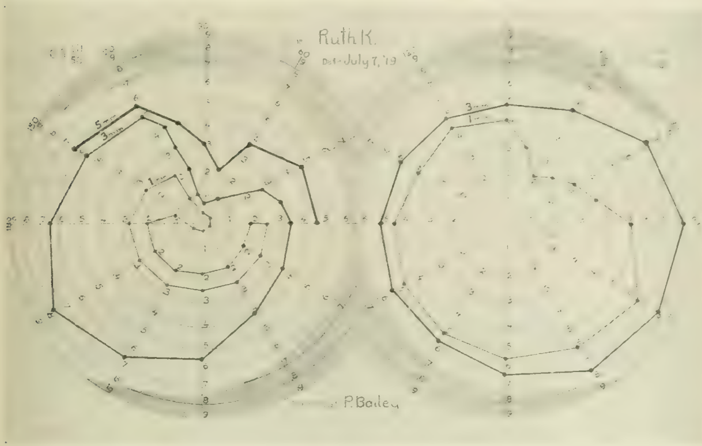


FIG. 47.—*Case* 10 (*contd.*). Field two weeks after negative exploration showing advance in process. (Cf. fig. 46.) On July 16 there was found a slightly more marked defect in the fields.

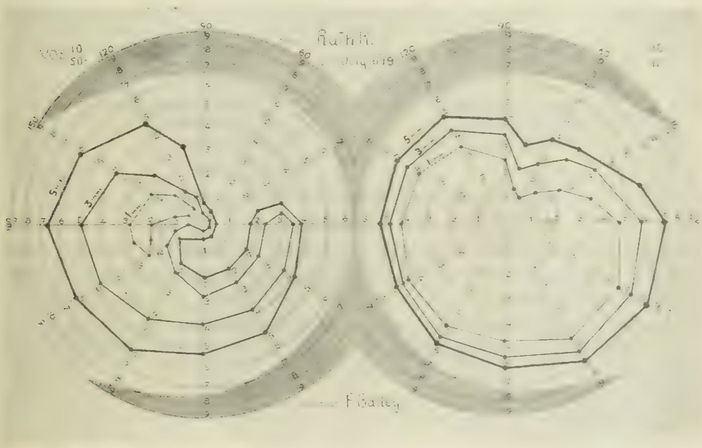


FIG. 48.—*Case* 10 (*contd.*). Fields three weeks after operation, showing further advance in defect.

On July 16 there was found a still more marked defect in the fields (fig. 48). The choked disc had largely subsided by this time and there was practically no protrusion of the decompression area. A week later, July 23, this peculiar field defect was still more advanced (fig. 49) and here it remained stationary.

There were grave doubts as to what course to pursue. An antero-posterior X-ray showed some absorption of the sphenoidal ridge and this made it seem probable that our original diagnosis had after all been correct and a tumour situated farther forward than anticipated had been overlooked. Her general condition had improved and the symptoms for the advancing field defect appeared to be stationary.

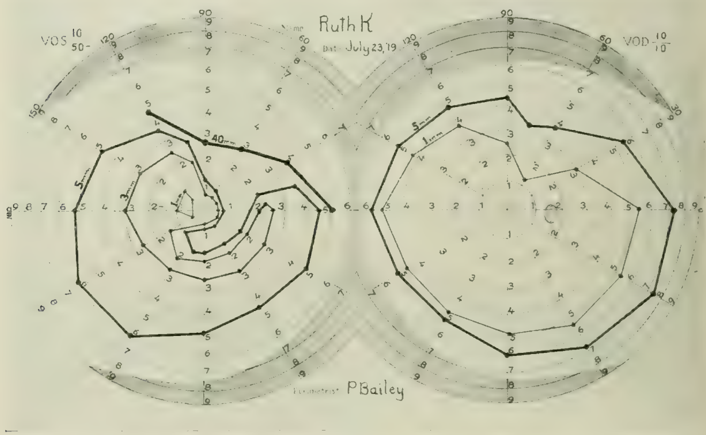


FIG. 49.—*Case* 10 (*contd.*). Fields one month after negative exploration, showing continued progress in the defect, chiefly of the left eye.

Most of the symptoms were so inconspicuous that no particular stress would have been laid upon them had it not been for the perimetric changes. They were in summary :—

Subjective : (1) Occasional numbness and discomfort of the left teeth.

(2) Occasional momentary tingling of the fingers of the right hand associated on two occasions with slight aphasia.

(3) Fleeting gustatory impressions.

Objective : (1) Slight exophthalmos of the left eye.

(2) Slight choking of left disc more than right.

(3) Upper right homonymous field defects.

August 11, 1919 : Operation 2. Partial enucleation of a large endothe-

lioma. A more thorough and more anterior exploration was carried out at this time. The margin of the tumour was brought into view. It had been covered and concealed by a thin shell of the temporal lobe. Its base of attachment was obviously at the sphenoidal ridge. The large growth was fragmented in removal and in the attempt to complete the enucleation there was bleeding and the remaining fragment had to be left in position. Even so, a cavernous sinus thrombosis was provoked which led to such a degree of proptosis that the eye, owing to keratitis, was finally enucleated on September 4. Normal field outlines were regained in the right eye by the time of her discharge.

One cannot be sure in a case of this kind (with a tumour compressing the lobe and a tumour, moreover, which arises from the meninges in such a situation that the optic tract itself may possibly have been pressed upon) whether the field distortion was actually due to involvement of the temporal loop. It, however, is more than likely, but in either case the diagnosis of a temporal lobe lesion was chiefly based on the advancing quadrantal defect. For the purposes of this paper, the case is important for the reason that the original fields of June 23 represent the earliest stage of such a defect that has been recorded in the series. To make a diagnosis of temporal lobe tumour largely on the basis of a notch in the upper nasal field in one eye would seem absurd were it not for the accumulated evidence regarding the localizing significance of these partial defects which this paper is intended to emphasize.

Temporal Lesions other than Tumour.

Though this communication deals primarily with tumour cases, it is not to be forgotten that the same principles hold true for lesions of other sorts. The illustration given at the outset, of a gunshot wound of the temporal lobe, is a case in point, and in all probability when the war material has been fully worked up, important contributions to our knowledge of temporal lobe symptomatology will be forthcoming, even though the urgency of military service in most hospitals for the recently wounded may have precluded such details as the use of the perimeter.

That the position of the radiation is poorly understood even when the perimeter has been used and a hemianopsia disclosed is evident enough, and in the minds of many physicians a homonymous hemianopsia indicates an occipital lobe lesion. I have even known of an occipital exploration for a shrapnel ball on this basis, conducted under the direction of a neurologist, when, as subsequent events showed, the missile actually was in the temporal lobe. Probably all of us have made

similar mistakes in tumour cases when the distractions of war could not be offered as an excuse.

But in civil life there are other sources of injury to the temporal lobe far more common than gunshot or stab wounds which may produce these field defects. Fractures of the base of the skull are possibly the most frequent source, for, as is well known, they are prone severely to contuse the tips of the temporal lobes. There are a number of cases of this sort in our records in which the perimeter has been employed with the disclosure of sharply cut sector-shaped defects (fig. 50). And I am

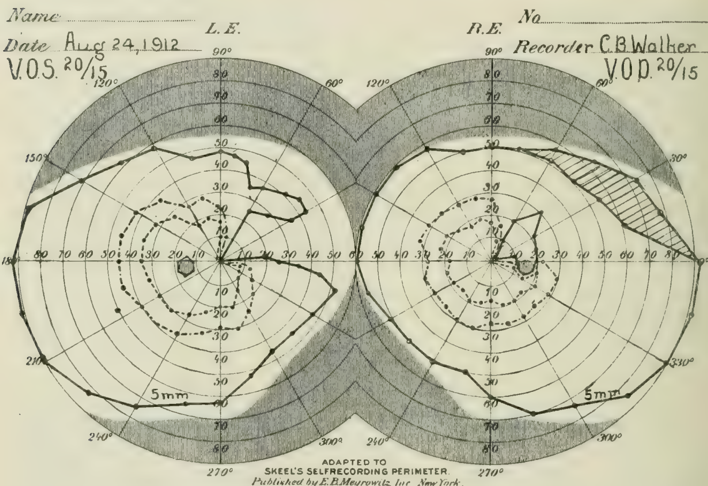


Fig. 50.—Sharply angulated homonymous field defects reaching to the central point following fracture of base of skull, with contusion by contrecoup of left temporal lobe.

under the impression that these traumatic lesions are more apt to produce defects in the fields characterized by sharp angulations than are tumours in whose presence the defects are ordinarily, if not always, rounded off. I have twice seen such defects as the result of the so-called prize-fighter's fracture of the base following a blow on the jaw transmitted through the condyle (fig. 51).

Another and still more important condition producing these defects is a temporal lobe abscess. Difficulty is often experienced in differentiating between a possible cerebellar or temporal lobe lesion when patients

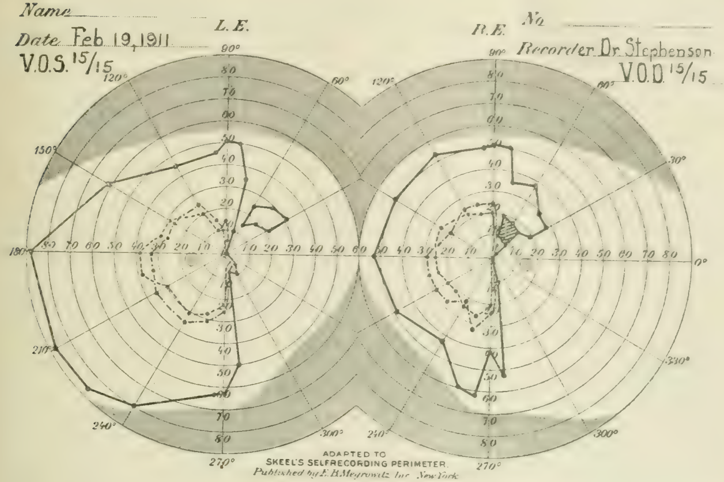


FIG. 51.—Incomplete homonymous hemianopsia following left temporal lobe lesion following a blow received in a boxing contest.

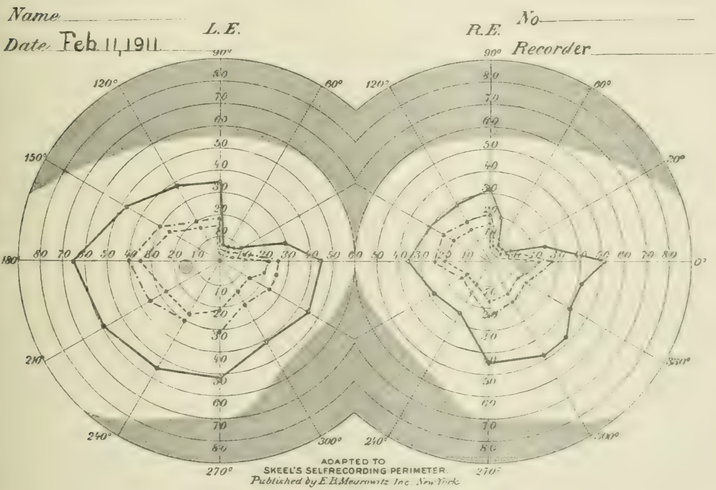


FIG. 52.—Right upper quadrantopsia in patient with left temporal lobe abscess.

are under the suspicion of having an abscess secondary to an otitis media. The perimeter, under these circumstances, may give invaluable information (fig. 52).

The General Symptomatology of Temporal Tumours.

It is but a scant twenty years since Byrom Bramwell, in an article on the localization of intracranial tumours [2], made the statement that tumours of the temporo-sphenoidal lobe and more particularly those of the right side were of all tumours the most difficult to diagnose and to locate, because these regions represented the most silent area of the brain. This statement by an eminent neurologist long interested in the special topic of brain tumours was utilized twelve years later by Foster Kennedy to introduce his article, which to the present time remains perhaps the most important single contribution (certainly in English) to the subject [4]. After referring to the comparative rarity of temporo-sphenoidal tumours Kennedy made a careful symptomatic analysis of nine verified cases from the records of the National Hospital at Queen Square. In conclusion he gave what he considered to be the essential symptom complex of these tumours for both right and left lobes. Common to a tumour of either lobe were: (1) convulsions of major and minor (uncinate) type, (2) bilateral choked disc, usually more marked on the side of the lesion, (3) post-epileptic transitory disturbances of motion and of the reflexes which later become persistent. In addition to these symptoms alike for both sides, a tumour on the left was said usually to be accompanied by some degree of aphasia.

It is evident from Kennedy's report that he was dealing with tumours of a more advanced grade than most of those which I have used as examples, for by the time an aphasia occurs in left-sided lesions together with weakness in the opposite face and marked change in the reflexes there is, in my opinion, considerable involvement of areas beyond the confines of the temporal lobe. It is, however, but natural, owing to the great advances in neurological surgery during the past decade, that many more of these lesions are to-day identified and at a much earlier stage than at the time of his paper. Hence in a considerable number of our verified cases, as indicated in some of those used in illustration (Cases 2 and 3 for example), the symptom complex pictured by Kennedy had not as yet appeared and the localization depended largely upon the field defects. The possibility of co-existent field defects was in Kennedy's mind, but he dismissed it with the following statement:—

"An examination of the pathological findings in my patients suggests the probability of hemianopia or hemiachromatopsia having been present during life owing to the proximity of the growth to the optic tract and radiations. In all cases the existence of hemianopic defect was considered and negatived as the result of examination. Five patients were examined perimetrically for various colours; the sole abnormality discovered was the concentric contraction of the visual field so often associated with severe, and especially with protracted papillœdema."

This quotation is given for the purpose of laying further emphasis on the value of the field defects in question, in the early diagnosis of temporal lobe lesions, rather than with any intent of pointing out an oversight of my distinguished predecessor.[1]

A tabulation of the symptoms recorded in the histories of the 59 verified temporal lobe tumours in my series has been prepared. As heretofore stated, in 6 only of the 39 cases in which the patient's condition made reliable perimetry possible were the fields normal at the time the tests were made. These were with two exceptions temporal endotheliomata which did not greatly deform the lobe and which in the absence of a choked disc probably would not have been detected at all had it not been for the unilateral exophthalmos and the overlying cranial hyperostosis, the usual tell-tale of these not uncommon tumours. The exceptions were: (1) a meningeal angioma; and (2) a glioma in which the recorded normality of the fields is perhaps subject to question. The fact, however, that field defects were present in 33 of 39 cases tested indicates that the perimeter, as a diagnostic aid in these lesions, is possibly the most important agent of all.

Though it was not my intent, in outlining this paper, to enter into a general discussion of temporal lobe symptomatology, the table which has been prepared to aid in this study brings out certain things in relation to the diagnosis which deserve to be briefly commented upon.

Generalized convulsions.—These were recorded in 20 of the 59 cases. They usually were few in number. A single convulsion without aura or sudden loss of consciousness, possibly unattended by convulsion, had been the inaugural symptom in three or four instances. A few of the patients had had only two or three convulsions and twelve was the largest recorded number.

[1] While this paper was going through the press my attention was called to an important article, "Die Tumoren des Schläfenlappens," by Albert Knapp, in the *Deutsche Zeitschr. f. d. g. Neurol. u. Psychiat.*, 1918, **43**, 226-89. In this elaborate study based on two cases Knapp mentions the fact that hemianopsia has been described. He does not lay any great stress upon this condition however.

The so-called uncinate seizures.—For the forty-five years since Hughlings Jackson's original description of these peculiar disturbances they have held the ground as the most distinctive and characteristic of all symptoms relating to the environment of the temporal lobe. This notable contribution to cerebral symptomatology, entirely consistent with the characteristics of its discoverer, was based on knowledge gained through animal experimentation subsequently transferred to the clinic and ultimately confirmed at the post-mortem table.

In the 59 cases of this series attacks often described as *petit mal*, or as more or less characteristic "dreamy states," were present in 24 cases. In 14 only of these cases, however, were there any olfactory or gustatory impressions and in some of these the possible uncinate character of the attacks was not particularly definite and appears to have been read into the patient's history after the lesion had been verified as a temporal one by operation. Nevertheless, when these Jacksonian seizures do occur in outspoken fashion they are unmistakable and of great diagnostic value, but, even so, speaking for the temporal lobe as a whole, they are far less common than are the field defects. Indeed, as might be expected of a lesion involving the uncinate gyrus, field defects were present with but one exception in every case having presumed uncinate seizures in which the use of the perimeter was not precluded by blindness or some other cause. On the other hand, field defects were present in eighteen cases in which no history suggestive of *petit mal* or dreamy states could be secured.[1]

Visual hallucinations.—Examples of these states have been given in the case histories (e.g., Case 6). Considerably to my surprise these visual pictures, often as vivid impressions, are recorded in thirteen out of the fifty-nine cases in the series. They, presumably, are part and parcel of the uncinate seizure, for in only three of the thirteen cases did they occur unrelated to what were regarded as attacks of this type. Hughlings Jackson, with Beevor, was among the first to describe this symptom [3] in a patient who had the impression of seeing "a little black woman engaged in cooking." A good example of the same thing is given by Kennedy in the history of his first case. These projection

[1] It is a matter of surprise to me that Hughlings Jackson did not emphasize the importance of the perimeter as well as of the ophthalmoscope, on which he laid such great stress in his writings (*Brain*, 1915, **38**, p. 391). The latter is unquestionably the more important instrument of the two, and as the profession was loath to adopt it, and still is for that matter, he may have felt that it was enough to urge its employment without forcing the perimeter on a reluctant profession at the same time.

pictures cannot be at all uncommon, and whether they are related in any way to the olfactory and gustatory discharges of the typical uncinate fit or are merely associated with some pressure implication of the visual pathway or the geniculate body, I am at a loss to say. Certainly they bear some relation to the damaged geniculo-calcarine radiation, for in this series, whenever its situation has been mentioned, the hallucination has always been referred by the patient to the side opposite that occupied by the lesion, in other words, in the defective fields.

One of the most remarkable examples of these hallucinations of which I have record occurred in one of the patients in my Baltimore series. It was a young man under observation for a long period who had repeated uncinate attacks, many of which were observed and described in detail. They invariably had the same character and left a vivid and disagreeable recollection. As in a dream he saw, always to his left side, his father and some other men in an unfamiliar room engaged in a game of cards which ultimately led to an altercation. The scene would fade and leave him with a bad taste and smell of something indescribably horrid. Objectively, during the course of ordinary conversation, possibly while standing, a vague look would come over his face, he would turn his head and stare to the left and shortly after would begin to make smacking and tasting movements with his lips. This was followed by an expression of disgust and the attack would be over. He had an homonymous upper quadrantal defect produced by a temporal lobe glioma.

One would naturally expect visual hallucinations to be a feature of occipital rather than of temporal lobe tumours, but the former are far less common in my series and, though they have not been thoroughly studied with this point in view, it is my impression that they are less prone to have *petit mal* attacks and that the subjective visual phenomena, if any, are more likely to be of colours and lights than of pictured scenes.[1]

[1] Though I have not fully looked up the general subject of visual hallucinations in this connection, it appears to me that those who have observed these states in association with a homonymous hemianopsia have taken it for granted, without any real justification, that the lesion necessarily was in the occipital lobe. This certainly is true of the case reported by Paul Camus (*L'Encéphale*, 1911, **6**, Part I, 521-531) and also of some of the other cases in the literature to which he refers. In an earlier paper by Pick (*Amer. J. M. Sc.*, 1904. **127**, 82-92) there is a good example of what I would take to be a left temporal tumour which he localizes, however, in the angular gyrus. All that can be said of such case reports is that without a post-mortem examination or verification of the situation of the lesion at operation, judging from the cases in this series, one at least is not justified in ascribing too much to the occipital lobe.

Auditory symptoms.—In view of the supposed relation of the temporal lobe and particularly of the transverse temporal gyrus to audition and the speech mechanism, it is extraordinary how slight and inconspicuous are the clinical evidences of any disturbance in this sphere. Certainly in none of the cases in this series has there been anything suggestive of auditory hallucinations of musical sounds or imagined speech, as might have been expected.

Without wishing to provoke a discussion regarding the location of the centres for the perception of speech, it has always been a matter difficult for me to understand why after a left subtemporal decompression for an unlocalizable tumour no disturbance of hearing occurs even though the lobe may bulge markedly through the defect. On the analogy of the paralyses which are known to result from the protrusion of cortex through a defect made over other parts of the brain, one would certainly expect some disturbance of hearing from a protrusion of the first temporal gyrus if this is as important an area as we have been led to believe.

In this series of 59 cases hearing was absolutely unimpaired in 38 of the patients. In eleven cases there was tinnitus heard in one or both ears but it was never a particularly constant or annoying symptom as in acoustic tumours. Slight contralateral deafness was recorded in five cases, slight relative deafness on the side of the lesion in two cases. In the remaining cases the tests were obscured by an old otitis media. To be sure, these tests were made merely by the voice, the watch and the fork and without any such accuracy of measurement as the perimeter gives for the fields of vision, but certainly, were contralateral deafness a common sequence of temporal tumours, occasional cases with pronounced loss of hearing would be encountered, and of these the records contain no example.

So far as aphasia is concerned there can be little question but that, when in left-sided cases a disturbance of speech is evident, one almost invariably can detect some evidence at the same time of a right facial weakness which indicates that the pressure effects of the tumour are exerting themselves on areas above the Sylvian fissure. I am a little in doubt, after re-reading Kennedy's paper, whether he regards the aphasia sometimes seen with left-sided temporal tumours as really temporal in origin. To my thinking, however valuable it may be in tumour lateralization, aphasia is a neighbourhood symptom and not a true temporal lobe phenomenon.

The temporal versus a cerebellar syndrome.—It would seem improbable that one could ever be misled by the symptoms of a temporal

lobe tumour into making the diagnosis of a subtentorial lesion. This nevertheless may occur, particularly in the absence of uncinate seizures and when field defects either are wanting or the perimeter cannot be used.

In fifteen of the fifty-nine cases there was definite nystagmus and in some of the patients it was a marked feature. Dizziness and vertigo are not unusual, and curiously enough primary suboccipital headaches have been fairly common. Then, too, in a few cases there has been definite ataxia and marked static instability. A few years ago one of my assistants, who had been devoting himself particularly to the cerebellar symptomatology and was an expert in the interpretation of Bárány tests, was led to make a suboccipital exploration in a patient who did not survive, and autopsy disclosed a temporal lobe tumour. By a sad oversight the fields of vision in this case had not been taken, for the cerebellar symptoms were supposed to be sufficiently definite to make this unnecessary.

It is not at all infrequent for my assistants or myself to make a tentative rating of a patient on the first superficial study as a cerebellar suspect and to have this presumptive diagnosis ruled out beyond question as soon as the fields have been taken. The chief difficulties arise when the patient is blind or uncooperative. It is of course one of the traditions of neuro-surgery that one may easily mistake a frontal for a cerebellar case or vice versa. This is well brought out by the tables of operations given in Tooth's studies of the National Hospital series, and I have twice been led to make a cerebral exploration for tumours as definitely localizable in the long run as are those of the acoustic nerve. Undoubtedly one must also bear in mind the possibility of confusing a temporal and cerebellar lesion, and it is safe to say that in every case of any obscurity whatsoever the perimeter should be employed.[1]

[1] In his article, "Die Tumoren des Schläfenlappens," by Albert Knapp (*op. cit.*), emphasis is also laid upon the difficulty of diagnosis between a temporal lobe and a cerebellar lesion. In his summary he says: "In the third place, of especial importance are the disturbances of equilibrium which are likely to be chiefly confused with cerebellar symptoms, and can be described as a pseudo-cerebellar temporal lobe ataxia. Less often there are other symptoms which are characteristic of affections of the posterior fossa, such as pain and stiffness in the neck, grinding of the teeth, corneal areflexa, nystagmus, and abducens palsy." He further adds that if these three things—oculomotor palsy, pressure against the cerebral peduncle, and inco-ordination—are present, the diagnosis of a temporal lobe tumour is assured. He evidently regards these three things as the essential feature of temporal tumours and the other symptoms of secondary importance, even disturbances of taste and smell.

SUMMARY AND CONCLUSION.

(1) The temporal lobe is a common seat of cerebral tumour (fifty-nine cases in a series of 276 verified supratentorial tumours).

(2) In the fifty-nine verified temporal lobe tumours, perimetry, owing to the advanced stage of the process, was precluded in twenty cases, but of the remaining thirty-nine homonymous field defects indicating involvement of the temporal loop of the optic radiation were present in thirty-three instances.

(3) Heretofore the most important symptom for temporal lobe localization has been the occurrence of the so-called uncinate fits, but in this series, even including as such all attacks of *petit mal* without gustatory impression, they have been recorded in twenty-four cases only.

(4) Visual hallucinations have been a frequent symptom of the temporal tumours in this series (thirteen out of the fifty-nine cases).

(5) Auditory phenomena in this series are conspicuous by their absence. In a few cases only has there been tinnitus and rarely some lowering of sound perception.

(6) The chief errors of diagnosis arise (i) when, with a total median hemianopsia, the occipital lobe is considered to be the tumour seat; (ii) in the absence of demonstrable field defects, when symptoms supposedly of cerebellar origin are to the fore.

Hence it is fair to conclude that perimetry gives us information of paramount diagnostic value, particularly in the early recognition of temporal lobe tumours, the partial field defects short of a hemianopsia being especially characteristic of involvement of the optic radiation in this region.

REFERENCES.

[1] ARCHAMBAULT, LA SALLE. "Inferior Longitudinal Bundle and the Geniculo-calcarine Fasciculus," *Albany Med. Ann.*, January, 1909.

[2] BRAMWELL, BYROM. *Brain*, 1899, **22**, 37.

[3] JACKSON, HUGHLINGS. *Ibid.*, 1889-90, **12**, p. 358.

[4] KENNEDY, FOSTER. "The Symptomatology of Temporosphenoidal Tumours," *Arch. Int. Med.*, 1911, **8**, 317-50.

[5] MEYER, ADOLF. "The Connections of the Occipital Lobes and the Present Status of the Cerebral Visual Affections," *Trans. Assoc. Amer. Physicians*, 1907.

[6] WALKER, C. B. "Colour Interlacing and Perimetry," *Trans. Amer. Ophthal. Soc.*, 1916, **14**, 684.

[7] *Idem.* "Quantitative Perimetry: Practical Devices and Errors," *Arch. Ophthal.*, 1917, **46**, 537-61.

[8] *Idem.* "Neurological Perimetry," *Trans. Ophth. Sect., Journ. Amer. Med. Assoc.*, June, 1917, 189-202.

REFLEX MOVEMENTS AND POSTURAL REACTIONS IN QUADRIPLEGIA AND HEMIPLEGIA, WITH ESPECIAL REFERENCE TO THOSE OF THE UPPER LIMB.

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CONTENTS.

INTRODUCTION.

SHERRINGTON'S original studies of the reflex reactions of the hind limbs in decerebrate and spinal animal preparations failed to excite much clinical interest until Babinski drew attention to the diagnostic significance of the plantar reflex in man.

This clinical discovery was indirectly responsible for a closer study of Sherrington's researches by neurologists, with the result that the reflex activities of the lower limbs in man have been the subject of many and important investigations. These have shown that the movement evoked by plantar stimulation is not an isolated phenomenon but forms a part of a general flexion or withdrawal action of the lower extremity. Its analogy with the phenomena described by Sherrington in sub-human movements under similar conditions and its presence in the healthy human infant gave it phylogenetic and ontogenetic significance.

The impetus given by these researches has led to widespread results. The questions of spasticity and other reflex reactions, as they occur in hemiplegia and paraplegia, have been reconsidered in the light of physiological experiments and much theoretical and practical knowledge has been gained in regard to the reflex behaviour of the lower limbs under normal and abnormal conditions.

Desultory attention only has hitherto been given to analogous reactions in the upper limb, largely owing to the rarity of cases of spastic quadriplegia of spinal origin.

Our interest in this subject was first excited in 1916 by the observation of a nociceptive reflex in the upper limb of a patient suffering from a high cervical injury and we have been fortunate enough to have had under our care five examples of quadriplegia showing reactions of this kind. Not unnaturally a search was made for similar phenomena in patients suffering from hemiplegia of cerebral origin and reactions presenting analogous features were frequently found.

It was soon apparent that the effects of stimulation of an upper or lower limb in both quadriplegia and hemiplegia were not confined to the stimulated part, but, under favourable conditions, were expressed as a generalized response in the somatic musculature.

Remote results were not found in response to artificial stimulation only but were evoked by strong voluntary efforts of the patients to make movements against resistance. A prolonged study of these widespread reactions has, we believe, thrown light on the whole question of the so-called "associated movements" which have long been recognized in connection with hemiplegic states.

The nature of muscular tone as a postural reaction had of necessity to be considered in relation to these problems, and numerous observations were made on our patients from this point of view.

As an introduction to our subject there will be described typical examples of quadriplegia and hemiplegia with analysis of the phenomena

they presented. Later chapters are devoted to a consideration of the relation of these phenomena to reflex reactions in decerebrate animals and to the theoretical conclusions which appear to be justified.

An appendix contains summarized records of some of the more important cases on which our paper is based.

CHAPTER I.—DESCRIPTION OF AN EXAMPLE OF SPASTIC QUADRIPLEGIA.

Case No. 1.—Charles V., aged 35, late Sergeant, Leicestershire Yeomanry, was wounded in the neck by a spent bullet on February 7, 1915. He was standing in a trench, leaning with his back against the parapet and talking to a sentry when he felt "something like a brick" hit him at the back of his head. He dropped immediately into the trench. Although dazed he remained conscious and heard the sentry call out "Sergeant V. is wounded." He remembers being placed on a stretcher and carried out of the trench. He was unable to speak for some time after he was hit and had considerable difficulty in breathing, being compelled to take quick, short breaths. He retains impressions of only a few disconnected incidents on his journey to the dressing station. According to his statement he was completely insensitive and paralysed, except for movements of his diaphragm, in his trunk and upper and lower extremities. Reflex micturition became established soon after his arrival at the dressing station and a catheter was passed once only, two days later, in the hospital at Boulogne. He arrived at 13th Stationary Hospital, Boulogne, on February 9, and we are indebted to Lieut.-Colonel Gordon Holmes for a note which he made on the patient's condition on the following day. A small, round wound was found one and a half inches above and one inch behind the tip of the right mastoid process. The patient declared that the bullet had been extracted from the collar of his coat but an exit wound was not discovered. There was no œdema of the scalp. The pupils were small but reacted well and no abnormality was found within the territory of the cranial nerves.

Motion: The patient could move his head. Feeble adduction at the shoulders was the only voluntary movement possible in the upper extremities. He was able to flex and extend his right lower extremity at hip and knee, dorsiflex, extend and invert his right foot and move his right great toe feebly. He was just able to move his left hip and knee but no other voluntary movement could be performed in that limb. In respiration his chest moved as a whole; it was uncertain whether the diaphragm contracted or not. The abdominal muscles were paralysed.

Tone: The shoulder muscles were slightly rigid, especially the pectorals in which the rigidity was constant; the muscles of the arm, forearm, and fingers were flaccid. The abdominal muscles were in a state of tonic contraction and the lower extremities were rigid, the left more than the right.

Reflexes: Arm-jerks were obtained, left brisker than right; the abdominal

reflexes were absent on both sides; the knee and ankle jerks were increased, and ankle clonus was evoked on the left side; the plantar reflexes were of the extensor type.

Sensation: There was complete loss of sensibility to pin-prick in the right upper extremity, but pricks were appreciated in the left arm below the level of the fourth cervical root distribution. On the trunk the area of complete cutaneous analgesia extended in front on the right side to within an inch of the nipple, and on the left side to the level of the top of the xiphoid. The loss to pin-prick was incomplete over the upper part of the chest and the neck up to and including the area of skin supplied by the third cervical roots. Appreciation of cotton wool was lost below the third cervical root area. The patient was unable to recognize changes in passive position of his left upper and ower extremities, but he responded well when his right lower and occasionally when his right upper extremities were tested. Vibrations of a large tuning-fork (C_{128}) were not appreciated on the upper extremities or below the level of the clavicles except on the right lower limb.

Condition on February 12, 1915 (five days after the injury): The patient complained of a tingling sensation when his left arm was touched, but no other change in his sensory state had occurred. The following reflex reaction was evoked: Stimulation of the skin on the inner aspect of the elbow by means of three or four pricks evoked a strong involuntary movement of the upper extremity consisting of internal rotation and adduction at the shoulder. Internal rotation was the strongest component of the movement. The reaction was obtained equally well in each upper limb but was strictly unilateral. The movement, which the patient was unable to initiate by voluntary effort, started unexpectedly after the stimulus was applied, developed rapidly, quickly subsided, and was very strong. The response was confined to the shoulder-joint, the pectorals and subscapularis appearing to be the chief if not the only muscles engaged. The receptive field comprised the whole inner aspect of the arm from the internal epicondyle to the axilla, and the reaction could be easily evoked by pricking or pinching the skin or by stroking firmly along the inner margin of the biceps.

On February 16 (nine days after the injury) the patient was evacuated to England and it was noted that voluntary power over the lower extremities had improved before his departure.

For the subsequent history up to the time when he first came under our observation we have to depend on the patient's account only. From the first he had never experienced the feeling that he was dissociated from his body and limbs, but when he shut his eyes he thought that his arms were flexed at the elbow, whereas in reality they were continually in tonic extension. His trunk and lower extremities seemed to him to be extended. About two months after he was wounded he was able to extend his right hand feebly at the wrist and to move his right thumb. A little later voluntary flexion of his right elbow became possible and within another fortnight movements began to return in his left upper limb in the same order as those on the right side. There was also some recovery of sensibility in his upper extremities.

He was able voluntarily to empty his bladder five weeks after the injury and about a week earlier could tell when his bladder and rectum were full. Within twelve months voluntary micturition was almost perfect and the rectal contents could be expelled, although he has continued to require aperients.

Erections of the penis occurred fairly frequently, but emission of semen had not been noticed since he was wounded.

Summary of his condition from August 1, 1918 (*forty-two months after the injury*) *when he first came under our observation* :—

He is a well-educated man and an exceptionally good witness. His mental capacity is above the average and his critical powers are excellent. Speech and articulation have never been impaired; there is no history of fits or convulsions and he rarely suffers from headaches.

The left pupil and palpebral fissure are smaller than those of the right side. The pupils react well to light and accommodation, but the dilatation to shade of the left pupil especially is slight. The cilio-spinal reflex is present on the left and absent on the right side. Ocular movements are full and there is no strabismus, nystagmus or diplopia. He wears glasses to correct a slight refractive error in both eyes (myopia and astigmatism) but his visual fields are full and optic discs and fundi normal. There is no evidence of abnormal function within the territory of the other cranial nerves.

Motion : Voluntary movements.—Voluntary movements of the head and neck are excellent and he can elevate his shoulders well. His right upper extremity is less paralysed than his left and all gross movements are fairly well performed. At the shoulder-joints adduction of the arm is a powerful movement and is much stronger than abduction. On the right side he is able to abduct his arm to an angle of about 90°, but on the left side the range of the movement is less. He finds it easier to move his arm backwards than forwards and internal rotation is performed much better than external rotation.

The movements at the elbow-joints are full in range; extension is distinctly stronger than flexion. He is able to pronate his forearms completely, but supination is a weak movement; on the right side the forearm can be rotated through an arc of about 130°, while on the left side it cannot be brought farther than the position midway betveen pronation and supination.

Extension and flexion of the hands and fingers are fairly well performed and flexion of the fingers is stronger than extension. Two years ago he was unable to execute any movement of an individual finger without bringing all the other fingers of the hand into action. This was particularly so on the left side. But as time went on isolated movements of the fingers and thumb of the right hand became more possible, although innervation still tends to be widespread, especially if he expends more effort than usual. With his right hand he can now unbutton his coat and arrange his cards at "bridge" while he holds them between his left thumb and index finger; but he is unable yet to button up his clothes or to write legibly. He feeds himself with his right hand but is obliged to have his food cut up for him.

Extension of the trunk is stronger than flexion. Movements of the chest have improved considerably but the diaphragm is still weak.

The lower extremities are stronger than the upper and the movements of the right lower limb are better than those of the left. Extension at hip, knee and ankle are stronger than flexion, and adduction at hip than abduction. The weakest movement is dorsiflexion with eversion of the foot. Movement of the toes is comparatively free.

All voluntary movements, especially those of the upper extremities, are performed slowly and stiffly, the reaction time being prolonged in relaxation as well as contraction. In attempting to grasp the observer's hand quickly it takes him a few seconds to bring his muscles into action and many more have passed before the maximum contraction of the flexors is attained. A similar delay is evident in the relaxation of his grasp. Co-ordination of muscles in the performance of voluntary movements is as good as could be expected in the presence of muscular rigidity.

Involuntary and synergic movements: An involuntary movement of the upper limbs not infrequently occurs in response to stimuli of widely different origin. A sudden emotional disturbance such as may be caused by the slamming of a door, a strong tickling sensation in his nose or irritation of his cornea by an eyelash evokes the following reaction which is often confined to his right upper limb but may involve both. The arms, rigidly extended, shoot out straight a little in front of his trunk and jerk forwards and backwards from the shoulders. The movement develops explosively, continues for a short time and subsides fairly quickly. The trunk and lower extremities apparently take no part in the reaction.

Rapid rhythmic flexion-extension movements of the thumbs at the metacarpo-phalangeal joints are of common occurrence, but we have been unable to trace the stimuli which excite them.

Flexor jerks of the right lower extremity sometimes disturb him in bed, but they have never been frequent. If he stretches out his lower limbs before getting out of bed in the morning, his right leg often becomes rigid in extension and shakes violently for a few seconds.

Vigorous voluntary muscular contractions in one limb are usually accompanied by movements in the others. For example, when the patient attempts to flex his right knee against resistance, his left upper limb may abduct slightly at the shoulder, flex at the elbow, extend at the wrist and metacarpo-phalangeal joints and flex at the interphalangeal joints, while the fingers and thumb spread out. In his right upper extremity there may be adduction, internal rotation, and backward movement at the shoulder, extension at elbow, pronation of the forearm, slight flexion of the hand, extension of the fingers, and adduction of the thumb. In his left lower extremity strong extension of the whole limb with inversion of the foot occurs. Similarly a voluntary attempt to extend the right upper limb at the elbow evokes flexion of the crossed upper limb and bilateral extension of the lower extremities. These associated movements will be considered in greater detail in the next chapter.

Tone: The attitude assumed by the patient in standing is the following (fig. 1). His lower extremities are fully extended at the knees with the feet about eight or ten inches apart. He stoops slightly forward with his shoulders

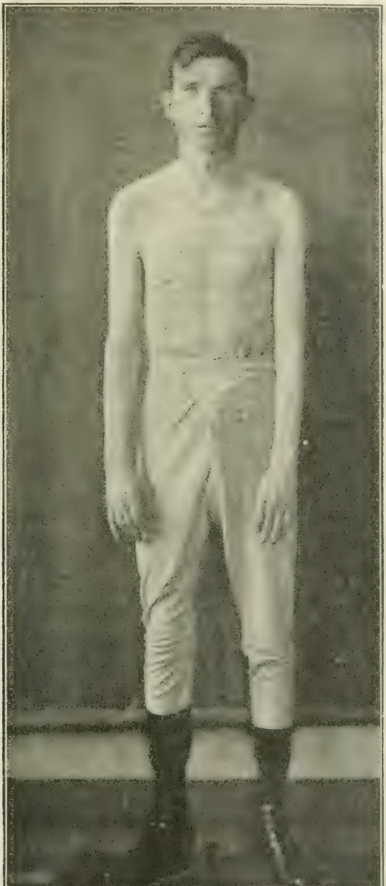

FIG. 1.—Photograph of No. 1 to show his general attitude in the erect position.

drooping, his arms a little in front of his trunk adducted and rotated inwards at the shoulders, rigidly extended at the elbows, the forearms pronated, the hands slightly flexed at the wrists, the fingers extended at the metacarpo-phalangeal

joints and flexed at the interphalangeal joints with hyperextension of the last phalanx of the thumb.

There is excessive rigidity of certain muscle-groups, especially in the upper extremities. The adductors of the arm, and in particular those of the anterior axillary wall, the pronators of the forearm and the flexors of the wrist and fingers, stand out prominently in tonic contraction and feel hard and tense when palpated. When muscle tonus is tested by the resistance offered to passive movement, it is found to be greatly increased in all the large muscles of the upper limbs, but in some more than others. The muscles with greatest tone are the adductors of the shoulders, triceps, the pronators, the flexors of the wrist and fingers, and the adductor-opponens group of the thumb. All the muscles of the trunk are hypertonic, but especially the erectores spinæ and the recti abdominales. The lower are less rigid than the upper limbs, but tonus is definitely increased, more so in the extensors than in the flexors. The left upper and lower extremities are more spastic than those of the right side.

Reflexes: In the upper extremities all the tendon jerks are increased. Any muscle when put on the stretch gives a brisk contraction in response to a tap on its tendon.

One of the interesting features in this case is a reflex reaction evoked by stimuli applied to the inner aspect of the upper extremity. Sensibility to pin-prick is defective along the inner aspect of the limb, yet when the skin in this region is firmly pinched the following response occurs explosively after a latent period of a few seconds. The arm suddenly shoots backward with extreme inward rotation, so that the palm of the hand faces almost directly outwards. There is adduction and internal rotation at the shoulder, extension at the elbow, pronation of the forearm, slight flexion at the wrist, and over-extension and adduction of the fingers. The fingers are so placed that they form a cone—the second finger overlapping the first and third, and the fourth underneath the third. The thumb is extended at the interphalangeal joint and adducted into the palm. The motor focus of the response is in the muscles of the shoulder-joint. When the reflex is excited while the patient is standing, the response is seen to involve the trunk and all four limbs, although it is most vigorous in the stimulated upper extremity. With extension, adduction, and inward rotation there is a backward thrust of the limb, the shoulder is pulled backwards, the upper part of the trunk rotates as a whole towards the stimulated side, and the patient is bent slightly forwards by the action of his abdominal muscles. The other upper limb becomes adducted at the shoulder and flexed at the elbow, but the movement is slight. The lower extremities remain extended at the knees and ankles.

The reaction outlasts the duration of a brief stimulus, so that the attitude provoked is maintained for many seconds and then gradually subsides, giving place to the original resting posture of the whole body.

One character common to all effective stimuli is the nocuous quality with which they are endowed. Pinching or scratching the palm of the hand, and the skin on the inner aspect of the limb and chest from the second to the

sixth costal cartilage are capable of evoking the response from either upper limb.

Reflexes are not obtained from stimulation of the upper or lower part of the abdominal wall, but can be elicited in the middle segment.

Cremasteric and bulbo-cavernosus reflexes are easily evoked on both sides.

Stimulation of the sole of the foot is followed by a typical flexion reflex consisting of flexion at hip and knee, dorsiflexion of the foot and upward movement of the toes. With a moderately strong reaction there is usually extension of the opposite lower extremity. The muscles of the abdominal wall do not always take part in the reaction. The receptive field for the flexion reflex extends from the plantar surface of the foot, where the threshold value of the stimulus is lowest, almost to the knee.

In response to stimulation of the skin or deep tissues of the thigh, extension at hip, knee and ankle and downward movement of the toes is usually obtained. If, however, the thigh is grasped suddenly and firmly, general flexion of the limb may result.

The knee-jerk on both sides is very brisk and with it there occurs adduction of the opposite thigh without flexion or extension of the hip.

The ankle-jerks are much increased and well-sustained ankle clonus can be evoked.

Sensation: The patient has been remarkably free from pain since his injury occurred. The only abnormal sensations of which he complains are numbness of the fingers, as if they were slightly swollen and cold, and "a feeling of tightness" in the muscles in front of his thighs, when he is bilious or out of sorts in any way.

Pin-pricks are appreciated as such, but with a raised threshold, over the post-axial aspect of the left upper extremity and hand, the pre-axial aspect of the right forearm and arm, and the shoulder and neck, below the level of the third cervical root distribution. On the right side of the trunk from the second rib to the groin pin-pricks are appreciated as points but fail to evoke definite pain. On the right lower extremity they give rise to a sensation of tickling only. Warmth is appreciated though with diminished acuity on the left side below the level of the third cervical root distribution. The defect is greatest on the lower extremity. Within the corresponding area on the right side the loss of sensibility to warmth is greater. Warmth is usually recognized correctly when the test objects are placed in contact with the upper limb and the chest, but below the costal margin the loss is complete. The loss of sensibility to cold is slight on the left side, but on the right side it is gross on the post-axial aspect of the upper limb and on the chest from the third to the seventh rib, below which level it is complete.

Tactile sensibility is not gravely disturbed except on the right side over the upper limb, the trunk below the second rib and the lower extremity. Complete loss to contact with cotton wool on hairless parts is limited to the right leg below the knee.

Vibrations of a large tuning-fork (C_{128}) are not appreciated on the trunk and

lower extremities below the first rib. On the upper extremities vibrations are appreciated only when the fork is vibrating well, but the defect diminishes towards the shoulder.

There is gross loss of recognition of passive position in all four limbs and the patient is entirely unable to recognize the shape, size and form of common objects placed in his hands. He has completely lost the power to estimate the differences in weights placed on his supported hands; tactile localization is disturbed less than the other spacial aspects of sensibility.

Vasomotor and nutritional changes: His skin is normal in colour and elasticity. There are no sores. In warm weather he sweats mainly from his head and neck and in these regions the hyperidrosis may be excessive.

Bladder, rectum and sexual functions: Control over the bladder is perfect. He is chronically constipated and requires to take aperients daily. Erections are frequent but there has been no emission of semen since the date of his injury.

Chapter II.—Analysis of the Reflex Phenomena in No. 1.

Posture: The attitude assumed by the body at rest is largely determined by the distribution of muscular tone. In the case of No. 1 the distribution of muscular tone was such that the posture of the trunk and limbs in the erect position or while recumbent was almost stereotyped; it varied within narrow limits only. In resting or sleeping he lay on his back with his head and shoulders slightly raised by pillows, his upper limbs lying by the side of his trunk in a position of general extension. The limbs were rotated inwards and adducted at the shoulders, extended at the elbows, with forearms pronated and wrists slightly flexed. His fingers were extended at the proximal joints and somewhat flexed at the interphalangeal joints; the first, second and third fingers were pressed together, while the fourth finger was a little abducted. The thumb, which was in apposition to the palmar surface of the index finger, was extended at the metacarpo-phalangeal joint and over-extended at the distal joint. His lower extremities usually lay extended on the bed, a little separated from one another, rotated outwards with the feet pointing.

The outstanding features of the general posture were the slight flexion of the neck and trunk from elevation of the head and shoulders, extension of all four limbs in line with the body and the strong inward rotation and pronation of the upper extremities. He was comfortable in this position for a long time and rarely moved much in his sleep. The extremities on the left side were more rigid than those on the

right and the upper more than the lower, so that if he desired to alter his position he did so by flexing his right lower limb and pushing with his foot against the bed, helping himself to some extent with his right arm.

But remarkable as was the patient's attitude when recumbent, the posture he assumed in standing was even more striking. The general features were the same in both positions, but the rigidity when erect gave him an almost statue-like appearance. This impression is illustrated by fig. 1.

The patient stood with his feet a little apart and his lower extremities fully extended at the knees; his trunk was slightly flexed with his head thrown back so that he could look directly forward. When he walked, however, he kept his eyes fixed on the ground a little in front of his toes. The photograph shows the strong tonic contraction of the upper fibres of trapezii, although the shoulders are drawn forward by the equally powerful contraction of pectorals. His upper limbs are rigidly extended and are adducted so that his hands lie in front of his thighs. They are turned inwards at the shoulders with the forearms pronated and the hands deviated somewhat radially at the wrists. The position of the fingers with extension at the metacarpo-phalangeal joints and flexion at the distal joints is well illustrated in the photograph. The thumbs are seen to be extended and pressed against the palmar surface of the first fingers.

Tone: The striking appearance of immobility of the patient when recumbent or erect was due to the excessive development of tone in his muscles, which, however, were not all equally loaded. The preponderance of tone in certain muscle groups was responsible for the constancy of the attitude of the body as a whole and of its component parts

The clinical tests at present employed in the investigation of the tonic state of muscles are crude and often untrustworthy, but in the case under consideration the distribution and certain other features of the rigidity were so distinct and constant that the following description can be accepted as fairly accurate.

All the muscles of the body from the shoulder-girdles downwards, with the exception of some of those of the fingers and toes to be detailed later, were in a state of increased tone. They felt firmer on palpation and presented more resistance to lateral displacement than the muscles of a healthy individual. The musculature of the extremities showed these features more on the left than on the right side, and that of the

upper extremities more than that of the lower. Detailed investigation revealed a greater increase of tone in some muscle-groups than in others. Thus in the upper extremities, and especially on the left side, the pectorals, triceps, pronators, and wrist-flexors were usually in a state of strong contraction. These muscles when palpated felt abnormally firm, and attempts to lengthen them passively were met by considerable resistance. Those of their antagonists, for example biceps and supinators which were readily accessible to direct examination, were found to be considerably less tonic and could be passively stretched with less difficulty.

The following tests brought out the difference in the tonic state of the different muscle-groups in another way. With the patient lying on his back the observer grasped one thigh and raised the limb from the bed. Instead of the knee flexing immediately as it would in a healthy individual the leg remained almost completely extended for a few seconds before the foot gradually sank towards the bed. Extension of the knee persisted for a longer time if the limb were lifted up quickly. It would seem that when the thigh was lifted the weight of the leg and foot excited an immediate reflex contraction of quadriceps cruris sufficiently strong and prolonged to keep the knee extended for some time against the influence of gravity.

This tonic rigidity of the lower limb in extension was increased and kept up for a longer time by evoking a flexion reflex in the opposite lower limb. For example, if the tonic reaction of the right quadriceps extensor was under observation and the point in the experiment was reached at which the leg began to fall towards the bed, complete extension could once more be obtained by scratching the sole of the left foot.

But, paradoxical though it may seem, it was possible to show that under certain conditions additional stimulation of the extensor reflex arc could temporarily decrease instead of augmenting the tonic extensor rigidity. The following experiment will illustrate the point: As before, the postural reaction in quadriceps extensor was evoked by suddenly raising the thigh from the bed, thus exciting the tonic reflex by mechanical tension. While the leg was being held in extension by contraction of the extensor of the knee, the patellar tendon was smartly tapped, thus supplying an additional brief stimulus of the same kind to the extensor reflex arc. This evoked a superadded contraction of the muscle in the form of a quick twitch, the foot first jerking upwards and then falling back again, but to a level lower than that at which it had been resting before the knee-jerk was excited. The jerk was

followed by three or four similar rapid movements of diminishing range before the leg finally came to rest. Although in the relaxation phase of each twitch the heel did not reach the bed, yet at the end of the reaction it was held considerably lower than at the beginning. In other words the knee instead of being in almost full extension was now somewhat flexed and complete flexion could be obtained as the end-effect of a series of knee-jerks.

Analogous reactions were obtained in triceps when the upper arm was passively supported in abduction and internal rotation, so that the weight of the forearm and hand pulled on triceps.

A different result, however, was obtained on investigation of the tonic reaction to gravity in the flexors of the elbow and knee. For example, in the case of the lower extremity with the patient lying on his face the leg was bent at the knee to almost a right angle, and then quickly released. Instead of being "held" by tonic contraction of the hamstrings in the position in which it had been passively placed it began almost immediately to extend and fell more rapidly than in the experiment with quadriceps extensor. So also with the flexors of the elbow, the tonic reaction excited by sudden tension on the muscles exerted by the weight of the limb was relatively much feebler than in triceps.

The unequal distribution of tonus was demonstrated in another way. As already mentioned, the voluntary movements which were most powerful were those in which the more hypertonic muscles were brought into action as prime movers. In the upper limbs these movements were adduction and internal rotation at the shoulder, extension at the elbow, pronation of the forearm and flexion of the hand and fingers; in the trunk extension and in the lower extremities extension and adduction at the hip, extension at the knee and downward movement of the foot. The muscles which determined these movements are exactly those which by their action determined the resting posture of the body. But not only were the movements just enumerated the most powerful, they were also performed most rapidly to command. Thus when on command the patient attempted quickly to abduct his arm, an interval of several seconds elapsed before the desired movement was begun, despite obvious efforts on the part of the patient to carry out the action promptly. The reaction time was long and the movement once started was slow and deliberate. The slowness of the response was all the more evident when for the performance of the movement it was first necessary to relax antagonists

which were in voluntary contraction. For instance, a longer interval elapsed between the giving of the command and the commencement of the movement of extension of the fingers when to carry out the action the patient had first to relax his grasp of the observer's hand, than when the fingers were primarily in the position of rest in slight flexion.

In contrast to the deliberate character of these movements was the greater rapidity with which those muscles that were overloaded with tone were voluntarily contracted. Thus the patient when asked to flex his fingers or adduct his arm carried out the movement comparatively quickly and with considerably less effort.

An outstanding feature of the rigid muscles was the way in which they reacted to passive lengthening. When the observer flexed the elbow, the patient meanwhile remaining entirely passive, and exerted a *steady* pressure throughout the movement, he became aware of an increasing resistance in triceps which developed fairly quickly to a maximum. This resistance was maintained at its height until flexion was almost complete and then rapidly declined. If the forearm was flexed through part of the arc only and was then released by the observer, extension at the elbow sometimes immediately followed. This reaction was due to the contraction of triceps which had been not only maintained, but considerably increased by the stimulus of passive lengthening of its fibres. Further observations, however, showed that the additional amount of tonicity caused by the mechanical tension on the muscle disappeared, and the new muscle-length was "accepted" provided that the tension stimuli were interrupted. Thus, when the elbow was suddenly flexed in short jerks by the observer the resistance offered by triceps was felt at the beginning of each movement to increase more rapidly than when flexion was carried out slowly. If an interval was allowed to elapse between each jerk, it could be readily ascertained by palpation of triceps, and by "sensing" the tension of the muscle by slight movement at the elbow, that the accretion of tone due to the sudden stretching of the fibres quickly disappeared. In other words, the muscle had assumed a new length and was being "held" by a tonic contraction of approximately the same strength as before it was disturbed by the passive movement. The muscle recovered its original tonic contraction for each new length. This phenomenon was evident over a wide range of different lengths of triceps, but was minimal or absent when the limb was in positions approximating complete flexion or extension at the elbow. The same phenomena

were evident in the majority of the other muscles belonging to the rigid group. Thus it was brought out by passive flexion of the knee and extension of the wrist. But in those muscles of this group in which tonicity was most developed, namely, the pectorals and pronators, the resistance encountered on steady passive stretching increased very rapidly, was maintained at its height throughout the process of displacement and did not appreciably diminish during the time that the limb-segment was being held in the new position by the observer. Further, unless the process of lengthening the muscles was carried out slowly the tension was apt to excite a powerful reflex movement of the limb as a whole, in which inward rotation and pronation with extension at the elbow formed outstanding components. With careful manipulation, however, the arm could be completely adducted, or the forearm completely supinated.

Examination of the group of muscles which exhibited a less degree of tonic contraction than those we have been studying showed that in them a lengthening reaction was inconstant and when present was in comparison poorly developed. When, for example, the arm was extended at the elbow by quick jerks, the resistance to the passive movement offered by the flexors was usually increased but to a less extent than with the triceps during passive flexion. Another point of difference was that the forearm had to be displaced through a larger arc before the muscular tension notably increased. The flexors of the elbow when passively lengthened appeared to become "gathered up" and to "set" in a new posture more slowly and less completely than the extensors.

It was possible to demonstrate that triceps and the extensors of the knee could accommodate themselves to new postures which involve shortening without appreciable diminution of tone. Thus, if the observer extended the elbow by moving the forearm with one hand while he grasped the triceps with the other, he was able to satisfy himself that, as the points of attachment of triceps were approximated, the muscle did not become flabby, but on the contrary remained firm to the touch. That is to say the tonic functions of triceps and quadriceps cruris were such that these muscles were maintained in approximately the same degree of contraction although the distance between their attachments was progressively diminished.

During a voluntary movement the muscles not concerned directly in bringing about the required displacement of the limb segment displayed certain changes. The antagonists relaxed and the contraction of the muscles concerned with the fixation of joints, necessary for the move-

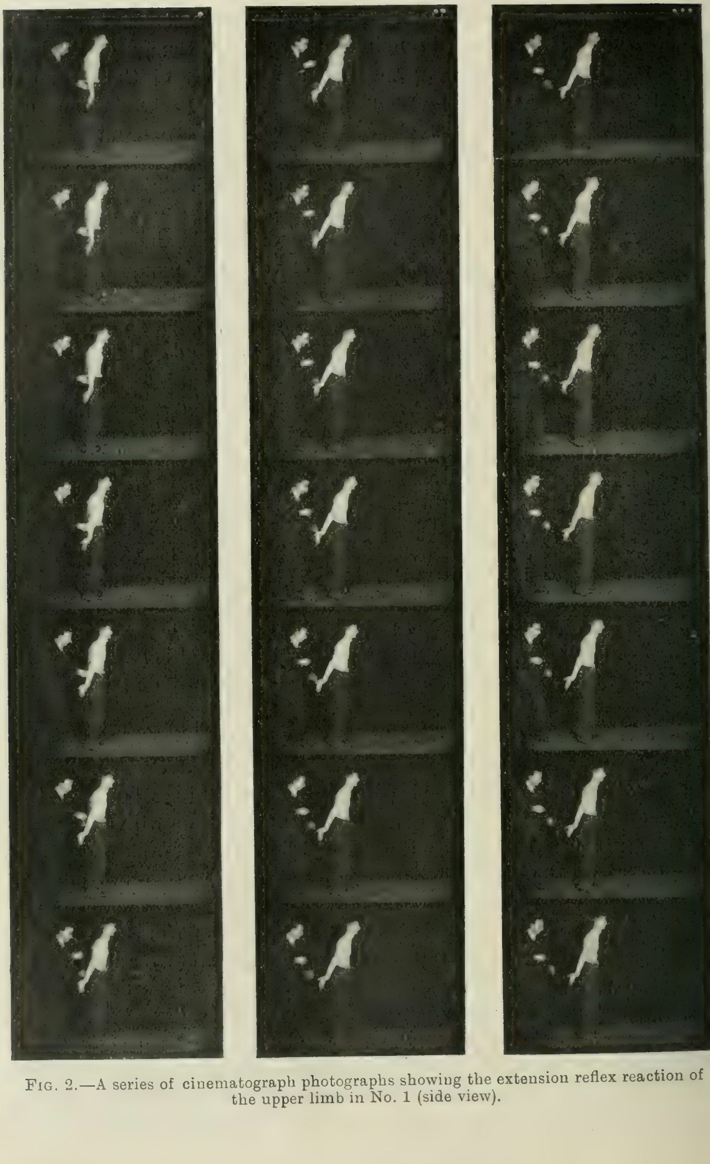

FIG. 2.—A series of cinematograph photographs showing the extension reflex reaction of the upper limb in No. 1 (side view).

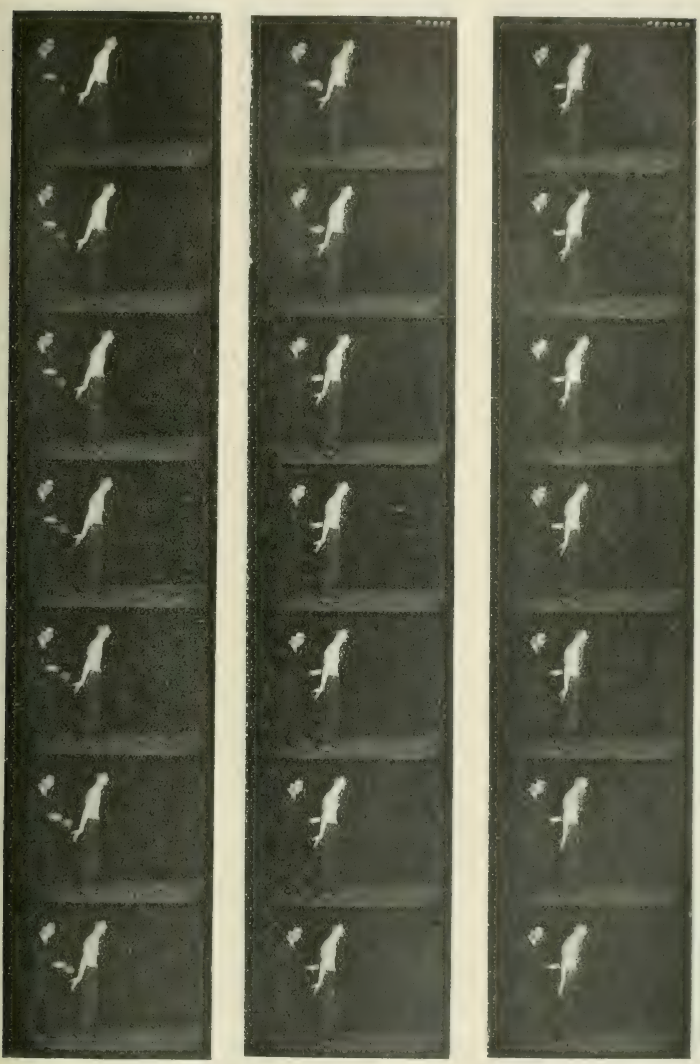

Fig. 2 continued.

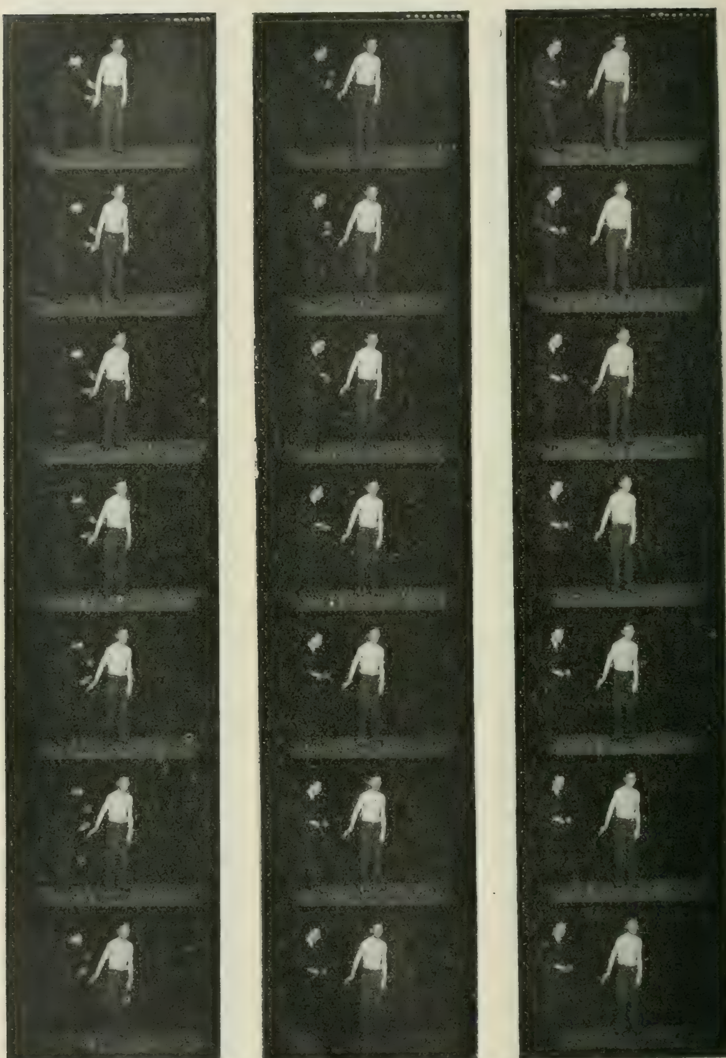

FIG. 3.—To show the extension reflex reaction of the upper limb in No. 1 (front view).

Fig. 3 continued.

ment, increased. If a strong voluntary effort was necessary to bring about the desired movement the tonic state of all the muscles of the limb might be altered. For example, when the patient was asked to squeeze an object firmly with his hand, all muscles playing on the wrist, elbow and shoulder-joints became more powerfully contracted; for the moment they were acting as fixators.

We have so far been dealing only with the tone of the large muscles of the trunk and extremities. The muscles of the toes, however, and certain of those which play on the finger joints have in this respect to be dealt with in a group by themselves. The toes and the fingers were freely movable in every direction and indeed the latter, especially the middle, ring and little fingers, could be hyperextended to an extreme degree without difficulty. The adductors of the thumb, however, were involved in the general rigidity and showed reactions to passive movement which were similar to those described for triceps, pronators and the other more hypertonic muscles.

Extension reflex of the upper limb. — Early in our investigations on No. 1 we discovered that an unusual and striking reflex reaction could be obtained in the upper extremity on either side in response to nocuous stimulation of the limb. With the patient lying in bed on his back the position of his upper limbs, it will be remembered, was one of adduction and inward rotation at the shoulder, extension at the elbow, pronation of the forearm and slight flexion at the wrist; the hands were not as a rule deviated to one or other side; the fingers were extended at the metacarpo-phalangeal joints and slightly flexed at the interphalangeal joints, while the thumb in extension was pressed against the palmar surface of the index finger.

On drawing the point of a pin or the tip of a finger-nail firmly along the ulnar aspect of the hand or forearm the following displacement of the limb occurred. There was strong adduction, internal rotation and retraction at the shoulder, extension at the elbow, extreme pronation of the forearm, increased flexion at the wrist, ulnar deviation of the hand, over-extension and adduction of the fingers and thumb. The fingers overlapped one another like the slates on a roof, the middle finger being posterior and lying over the index and ring fingers, while the thumb with extended phalanges was tucked into the palm and more or less in line with the middle finger.

The reaction could be observed more completely when the patient was standing and, in general, consisted of a vigorous movement by which the upper limb was adducted, extended and thrust backwards

with extreme inward rotation of the limb as a whole, so that the palm of the hand was directed backwards and outwards (figs. 2 and 3).

Although certain of the muscles involved could be seen and felt to contract almost immediately on application of the stimulus, usually an appreciable length of time elapsed before the movement took place. It was as if the impulses excited by the stimulus had to break through a strong resistance or had to reach a certain maximum intensity before they were capable of evoking the response in full. This supposition gains support from the fact that a brief harmful stimulus was as a rule ineffective; a prolonged stimulus was often required. For example, a single prick usually produced very little effect; the point of the pin had to be dragged across the skin and, although the pectorals contracted almost from the commencement of stimulation, the distance traversed by the pin often reached two or three inches before the limb was displaced. But once the point of sufficient intensity of stimulation was reached the reaction, although it could be graduated to some extent, tended often to occur explosively and the movement was sometimes so violent as almost to throw the patient off his feet.

Relaxation of the contracted muscles did not immediately follow the full displacement of the limb (figs. 4 and 5). The new position was maintained for several seconds and then the limb slowly resumed its original posture at rest.

Usually the reflex was experimentally excited with the upper extremity in the resting attitude of extension, adduction and pronation; but investigations showed that alteration of the initial position of the limb, for example flexion at elbow carried out either by the observer or by the voluntary effort of the patient, did not lead to any material change in the response.

A point of considerable interest is that the patient seemed to have little power to inhibit or control the reaction.

The muscles mainly concerned in bringing about the movement were those which were overloaded with tone and in consequence determined the attitude of the limb at rest. Incidentally attention may again be drawn to the fact that it was exactly those muscles which were rigid and which acted as prime-movers in the extension reflex that could voluntarily be brought into action most strongly and readily by the patient.

The condition of the antagonists in the reaction was noteworthy. Deltoid, biceps and supinator longus from their superficial position could readily be investigated by palpation. They were certainly actively

employed in the reflex when it was fully excited; they became firmer and could be transversely displaced less easily although their tonic contraction was less than that of the prime movers. Even by the rough methods of testing employed it was also possible to satisfy ourselves that the increase in tone of biceps was proportionate to the increased contraction of triceps. In other words, there was a definite relation between the state of the two opposing muscles. It must be clearly understood, however, that these statements hold good for the period of

FIG. 4.—Photograph showing the attitude of the right upper limb at the height of the extension reflex reaction in No. 1 (side view).

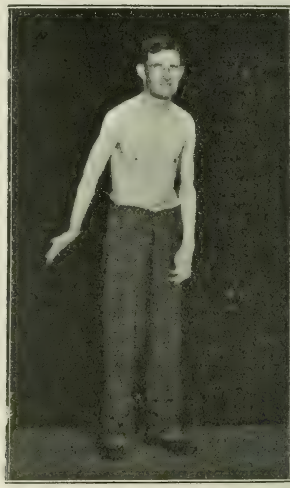

FIG. 5.—Photograph showing the attitude of the right upper limb at the height of the extension reflex reaction in No. 1 (front view).

time only in which the new posture of the limb was being maintained. It was impossible by our methods to ascertain the tonic state of the antagonists whilst the arm was being displaced in one or other direction by the reflex.

The receptive field of the reflex was extensive, comprising almost the whole limb and invading the chest in front and behind. The part of the field in which the threshold value of stimulus was lowest was the skin covering the walls of the axilla, but the threshold was not greatly higher for the inner aspect of the arm, forearm and palm of hand.

Within this area a moderately firm scratch with a sharp object or a prolonged squeeze of the skin or deep tissues formed an appropriate stimulus. The reflex was readily excited by pinching the tendons of biceps, pectoralis major or latissimus dorsi or by attempting suddenly to rotate the arm outwards at the shoulder or to supinate the forearm.

A response was never obtained from stimulation of the dorsum of the hand or fingers but was occasionally elicited when the stimulus was applied to the skin on the outer aspect of the limb. The receptive field on the front of the chest covered an area which extended from about the third to the sixth ribs, and on the back roughly corresponded in size and position to that on the front. Above and below these levels we were never able to excite the reflex with the stimuli we employed.

The reaction varied within narrow limits with the situation of the stimulus. Thus when the reflex was excited from the palm, movements of the fingers occurred early in the experiment and before the general reaction was evoked. Most often the preliminary movement consisted of extension and abduction of the fingers and thumb, while in the general reaction the digits were strongly adducted. Pronation of the forearm was a prominent early movement when the site of the stimulus was on the ulnar aspect of the hand or forearm, and diminution of the pronated resting attitude, usually with abduction of the thumb, followed application of the stimulus to the thenar eminence. The outstanding early movements which resulted from stimulation of the inner aspect of the upper arm or the chest were adduction and internal rotation at the shoulder and extension at the elbow. Indeed, from whatever point within the receptive field the reflex was evoked the muscles of the anterior axillary wall could almost invariably be seen to contract early. It may be remembered that attention was drawn on p. 400 to the response obtained by Lieut.-Colonel Gordon Holmes five days after the injury. Nocuous stimulation of the skin on the inner aspect of the arm between the axilla and elbow evoked a strong involuntary movement of internal rotation and adduction at the shoulder; at this time no movement at the elbow was observed. He especially noted that inward rotation of the arm was the chief component of the movement and predominated over adduction. This movement has remained the minimal response for the whole period during which the patient has been under our observation.

Two noteworthy variations in the reaction were sometimes observed by us when the appropriate stimulus was of a special kind and applied in the neighbourhood of the shoulder. While investigating by passive

movements the tonic state of the internal rotators at the shoulder a reflex movement of the whole upper limb was often excited, especially if the observer suddenly stretched the muscles. At times the movement consisted of the adduction-pronation-extension response already described, but often the reaction was more complicated. What happened was briefly as follows. When the arm was rotated quickly outwards the resistance encountered steadily increased if the attempt to overcome it was persisted in. The suddenly applied tension stimulus had succeeded in evoking superadded contraction of the internal rotators, the strength of which increased proportionately to the strength of the stimulus. With internal rotation at the shoulder there occurred flexion at the elbow and wrist, flexion and abduction of the fingers and extension and abduction of the thumb. This posture was broken into from time to time and replaced for short periods by extension of the elbow, over-extension and adduction of the fingers and adduction of the thumb.

The second pronounced alteration in the extension reaction was sometimes observed when the reflex was evoked by pinching the muscles of the anterior or posterior axillary walls. It consisted of a rapid, rhythmic, flexion-extension movement at the wrist instead of the more usual maintained attitude of flexion. The reaction at the shoulder, elbow, finger and thumb joints did not, however, differ from that obtained by stimulation elsewhere.

There were no essential differences in the reflexes excited in the two upper extremities.

Motor phenomena accessory to the extension reflex of the upper limb.—As a rule the motor effects of the reflex spread beyond the stimulated upper limb and involved the trunk and other three limbs. The trunk and lower limb on the same side were affected as soon as or earlier than the other upper limb. The associated reaction consisted of (1) contraction of all the muscles of the trunk and of the three limbs—those of the shoulder and hip joints being first involved, and (2) movements which usually were slow and deliberate and were due to greater contraction of some muscles than of their antagonists. The most common associated displacements were slight adduction and external rotation at the shoulder, flexion at the elbow and slight extension at the wrist in the opposite upper limb; the trunk was usually rotated and became slightly flexed and the lower limbs remained extended at the knees and ankles. The serial cinematograph photographs on pp. 412 to 415 represent a general reaction of this kind.

Motor phenomena in association with the flexion reflex of the lower extremity.—Stimulation of the sole of the foot (pinching, pricking or scratching) evoked a typical flexion reflex of the lower extremity consisting of flexion at hip and knee, dorsiflexion at ankle and upward movement of toes. The plantar aspect of the foot formed the focus of the receptive field which, for stimuli of moderate intensity, reached as high as the knee. Stimulation of the thigh as a rule evoked extension at hip and knee and downward movement of the foot and toes, but if the stimulus was intense (firm squeezing of the thigh) flexion of the limb frequently formed the primary response. Contraction of all the limb-extensors was readily excited by sudden passive dorsiflexion of the foot.

The strength of the flexion reflex could be varied to some extent by grading the intensity of stimulus, and the motor effect in a mild reaction was usually confined to the stimulated lower extremity. But by intensifying the appropriate stimulus it was possible to obtain a more widespread reaction the extent of which could also be controlled more or less by regulating the strength of stimulus. In the order of spread of the reflex the opposite lower extremity was first affected, then the muscles of the abdominal wall and back and lastly the upper limbs.

In the associated reaction all the muscles acting on a joint were contracted, but usually some more strongly than others, so that movements resulted. Movements, however, were sometimes absent, especially in weak reactions, and when present were never excessive and developed slowly. The crossed lower limb remained extended at hip and knee and the foot and toes moved down. The upper limbs became rotated outwards at the shoulders, flexed at the elbows, extended at the wrists with slight supination of the forearms; the fingers became extended at the metacarpo-phalangeal joints and flexed at the interphalangeal joints while the thumbs were extended and abducted. The muscles of the proximal joints of the limbs were always affected earlier than those nearer the periphery of the limbs. If the stimulus was weak the upper and lower limbs almost immediately resumed their resting attitudes, but in a strong reaction there was a definite after-action so that the postures were maintained for a short time before the limbs slowly resumed their resting positions.

Motor phenomena in association with voluntary movement of one limb against resistance.—Voluntary efforts of the patient to move one of his limbs or a segment of it when strongly resisted by the observer as a rule produced alterations in the attitudes of some or all of the other

limbs. These adjustments were not constantly the same on different occasions for any one voluntary movement. As with the general reactions associated with the extension reflex of the upper limb and the flexion reflex of the lower limb, the essential feature of the response was increased contraction of antagonists as well as prime movers so that movements when present were slow.

With forced voluntary movements of flexion or extension of the right lower extremity the response in the left was almost invariable extension. If at the beginning of the experiment the patient was lying in bed with his lower limbs stretched out, the only segments of the left lower limb which moved were the foot and the toes and these were thrust downwards, but the flexors as well as the extensors of the knee and ankle were in strong contraction, the extensors predominating. In a strong reaction extension of the crossed limb was accompanied by internal rotation at the hip.

The response in the right lower extremity in association with forced voluntary flexion or extension movements of the left lower limb differed as a rule in certain particulars from the reaction which has just been described. The want of correspondence may have a definite relationship with difference in the development of resting tonus in the muscles of the two limbs; the left was more rigid in extension than the right. The main points of divergence lay in the reactions at hip and knee, for when the right was the crossed limb some degree of flexion was usually observed. It must be understood, however, that although slight flexion occurred in the early phase of the response the movement formed a minor part only of the whole reaction which comprised a tonic contraction of all the muscles of the limb.

The movements and postures in the upper limbs induced by the voluntary efforts of the patient to move one lower extremity against resistance were subject to greater variation than those we have just studied in the crossed lower extremity. The principal variation was in the direction of the movement at the elbow, but it did not seem to occur in one limb more than in the other; neither did it apparently depend on which lower limb the patient was attempting to move nor on the direction of the movement, whether flexion, extension, adduction or abduction. The reactions in the upper limbs were also feebler than the response in the crossed lower limb. Three main reflex-figures were observed in the upper limbs: (1) Internal rotation and adduction at shoulder with extension at elbow on the side of the lower extremity

which was being voluntarily displaced by the patient, and external rotation at shoulder and flexion at elbow in the upper limb on the opposite side. (2) Bilateral adduction at shoulder, extension at elbow and wrist with extension and abduction of the fingers at the metacarpophalangeal joints, and flexion at interphalangeal joints and abduction of thumb (the so-called "claw" position of the hand). (3) The same posture in both upper limbs as last described except that the elbows were slightly bent.

With forced voluntary movements of the upper limbs the lower extremities invariably remained in extension, although the tonic contraction of all their muscles was increased. The response was stronger in the lower limb on the same side and here extension was often accompanied by adduction and inward rotation.

In contra-distinction to what obtained in the lower limbs the direction of voluntary displacement of the forearm usually altered the posture of the opposite upper limb at the elbow. Thus forced extension of the elbow on one side was accompanied by flexion of the elbow on the other side; the movements on the two sides were usually of opposite direction.

In each general response all the muscles of the trunk were brought into action.

As has already been pointed out, all the associated reactions which have been described in this section were made up of two components: (1) the maintenance of attitudes or postures due to the tonic activity of prime movers and antagonists; and (2) displacement of segments of the three limbs brought about by predominant contraction of certain muscle-groups.

Involuntary movements of the upper limbs excited by stimulation of parts outside the paralysed regions of the body and by changes in the patient's mental state.—Sometimes as the patient lay quietly in bed or was sitting up in a chair his arms suddenly shot out in front of him, extended at elbow, wrist and finger joints, and flapped up and down from the shoulders for several seconds. The occurrence of these involuntary movements could not be traced to irritation of the limbs or trunk or to voluntary effort on the part of the patient, and we were at a loss how to explain them. The patient, however, soon noticed that there was a direct relationship between them and the development of a sensation of tickling in his nose, or a sensation of irritation of the surface of his eyeball, such as is caused by an inverted hair of the lower lid. He was particularly subject to disagreeable sensations in his

nose so that the rhythmic movements of his upper limbs occurred frequently.

These involuntary movements were, however, evoked not only by stimulation of the nostrils and eyeballs, but also frequently followed a sudden emotional disturbance. For example, if the patient was suddenly startled by the slamming of a door or by some other cause his upper limbs were immediately thrust out and began to move up and down from the shoulder. The movements never continued for long at a time but he had little power to control them.

Chapter III.—An Example of Spastic Hemiplegia.

Case No. 5.—A. C., aged 41, house painter. At the age of 18 years he developed a sore on his penis, which was untreated. He married at the age of 21 and his wife has borne him three healthy children. There is no history of miscarriages. Nine years ago the patient was treated in the Venereal Department of Guy's Hospital for an ulcer on his tongue. On Easter Sunday, 1919, while out walking, he was suddenly seized with severe pain in his right wrist and shin. It passed off in a few seconds and he was able to walk home. When he awoke the next morning he found that the right half of his body was paralysed and he was unable to speak.

He received a course of anti-syphilitic treatment in Guy's Hospital in May and June, and he recovered sufficiently to be able to walk with the help of a stick. In November, 1919, he came under our observation and presented the following features.

He was a fairly intelligent man with a good memory. Speech was defective, but he could answer questions and express his thoughts in speech if given time. In the evenings he complained of a sensation of freezing in the left side of his head, but he was free from headaches. He did not suffer from fits. His visual fields were full and his fundi normal. He showed weakness of voluntary movement on the right side of his face; his tongue when protruded deviated to the right, and his jaw and palate movements were weak on the right side, but associated facial movements were stronger on the right than on the left side. Otherwise there was no defect in the functions of the cranial nerves.

All voluntary movements of his right upper extremity were weak, those of the distal segments being most affected. Abduction of the shoulder, flexion of the elbow, pronation and supination of the forearm, extension of the wrist and fingers were particularly weak and voluntary movements of the individual fingers were impossible.

His right lower extremity was stronger than the right upper limb; voluntary extension and adduction of the hip, extension of the knee and downward movement of the foot were powerful and considerably stronger than the other movements of the limb.

The voluntary movements of the left side of his face and of the left upper and lower extremities were unaffected.

The tendon jerks of the upper and lower limbs on the right side were brisker than those on the left. The right abdominal reflexes were diminished in the upper and middle segments and absent in the lower. Cremasteric reflexes were obtained on both sides. The right plantar reflex consisted of upward movement of the toes and foot and flexion of the knee and hip. This reaction was obtained by pinching any part of the leg below the knee as well as by scratching or squeezing the foot. The threshold of stimulus was lowest in the sole of the foot. No associated movement in the opposite lower limb was observed, but flexion of the left knee was evoked on several occasions by squeezing the muscles of the right thigh. The minimal response to plantar stimulation on the left side was downward movement of the toes and contraction of tibialis anticus; a stronger stimulus evoked, in addition, upward movement of the foot and flexion of the knee and hip, and in the opposite lower limb contraction of the extensors of the hip and knee and downward movement of the foot and toes.

Sensibility to light touch, pin-prick, thermal stimuli and changes in passive position were not diminished, and recognition of size, shape and form was as good with the right as with the left hand. The threshold for pain evoked by deep pressure (algometer) was lower on the affected than on the normal hand. Vibrations of a tuning-fork (C_{128}) were appreciated better on the left than the right side.

At first he had slight difficulty in starting micturition, but this disappeared later. Aperients were often required to overcome constipation.

Vasomotor, sudorific and nutritional changes were not observed.

Posture.—The right upper limb was slightly abducted and internally rotated at the shoulder, flexed 45° at the elbow, three-quarters pronated, hand in line with forearm, thumb extended at metacarpo-phalangeal joint and slightly flexed at interphalangeal joint, and fingers three-quarters flexed at all joints.

The right lower limb was slightly rotated outwards at hip and extended at all joints with the foot turned slightly inwards.

When the patient was standing a slight droop of the right shoulder was noticeable in spite of the fact that trapezius on that side could be seen to stand out in strong contraction. At the same time the attitude adopted by the trunk was one of slight flexion.

Tone in the right upper limb was generally increased but distinctly more so in the adductors of the shoulder, the flexors of the elbow, the pronators of the forearm, and the flexors of the wrist and fingers than in their respective antagonists. There was less resistance to passive flexion of the fingers than to movements at any other joint. In the lower limb tone was relatively greater in the extensors than the flexors, in the adductors of the thigh than in the abductors, and in the invertors of the foot than the evertors. In the trunk the extensors were on the whole more loaded with tone than the flexors.

The phenomena associated with increased tone, which we have already described in relation to our quadriplegic patient in the last chapter, were mani-

fest in the case of hemiplegia under consideration as well as in others under our observation. There is nothing to add to our description on p. 408 of the shortening and lengthening reactions, and the responses to the tests employed had, in general, the same features.

Flexion reflex of upper limb.—When the palm of the hand was scratched or pinched, the following movements were observed : Slight flexion of the fingers and wrist, more powerful flexion of the elbow, usually pronation of the forearm, abduction and external rotation and elevation of the shoulder. The threshold of stimulus was lowest in the palm of the hand, but a response could be easily obtained by scratching or pinching the skin on the inner aspect of the arm and forearm or that on the chest, behind and in front, corresponding roughly to the distribution of the third and fourth thoracic segments.

Reciprocal relaxation of the adductors (pectorals) accompanied contraction of the abductors and external rotators of the shoulder, including latissimus dorsi. With contraction of biceps there was usually relaxation of triceps.

The response varied slightly in detail with the locality of the stimulus. Thus scratching the radial side of the palm evoked abduction with extension of the thumb and some supination of the forearm. When the stimulus was applied to the inner aspect of the forearm near the elbow flexion of that joint was exaggerated, and the same observation applied to external rotation and abduction of the shoulder when the skin of the anterior wall of the axilla was pinched. Elevation and retraction of the shoulder were often observed following stimulation of any part of the reflexogenous zone.

The influence of the locality and duration of the stimulus were well shown by the response evoked by drawing the point of a pin from the elbow to the palm of the hand along the inner aspect of the limb. The response began with abduction, retraction, and elevation of the shoulder and flexion of the elbow ; as the stimulus approached the wrist, this joint became flexed, and when the palm was reached a movement of flexion of the fingers appeared. The response continued so long as the limb was being stimulated ; with removal of the stimulus the muscles gradually relaxed.

Single pricks, when effective, evoked a momentary quick response.

This reflex movement was more deliberate and less explosive in character than the extension reaction of the upper limb in quadriplegia and, as sometimes seen, in hemiplegia (No. 8).

Motor phenomena in association with the flexion reflex of the right upper limb.—With strong reflex flexion of the right upper limb there occurred contraction of deltoid, pectorals, triceps, and possibly biceps of the left side, although no displacement of the left upper limb actually occurred. All the muscles of the trunk on both sides were contracted. In the lower limbs all the muscles acting on hips, knees, and ankles became strongly contracted, those on the right more than those on the left side. Both remained extended.

Motor phenomena in association with the flexion reflex of the right lower limb.—All the muscles of the left lower limb became contracted, but the limb remained firmly extended. The trunk muscles were called into action on both

sides in front and behind. In the right upper limb there was strong contraction of the abductors and adductors of the shoulder; of the flexors and extensors of the elbow with a slow and incomplete movement of extension; of the flexors and extensors at the wrist with extension at this joint; and of the flexors of the fingers so that the hand became clenched. Sometimes rapid movements of extension alternating with flexion of the wrist were observed. In the left upper limb both adductors and abductors of the shoulder and flexors and extensors of the elbow were brought into action, but no displacement of the limb could be detected.

Motor phenomena in association with voluntary movements of one limb.— With powerful voluntary flexion or extension of the left elbow against resistance a strong reaction occurred in the right upper limb, consisting of increased contraction of all the muscles, but of some more than others, with the result that the following displacements occurred: Adduction and internal rotation of the shoulder, incomplete extension of the elbow, strong extension of the wrist with complete flexion of the fingers. Alternating flexion and extension movements at the wrist were sometimes noticed. All the muscles of the trunk were brought into action, the extensors more strongly than the flexors. There was bilateral extension of both lower limbs with increased contraction of all the muscles. The reaction was more powerful on the right side, where it was accompanied by inversion of the foot.

When the right elbow, that of the paralysed side, was voluntarily flexed against resistance, the reaction in the lower limbs was the same as that just described. The response in the crossed (left) upper limb was feeble in comparison with that obtained in the right upper limb, when the left (non-paralysed) upper limb was the seat of voluntary effort. It was confined to the proximal joints, and included slight flexion instead of extension of the elbow.

With voluntary flexion or extension of the left knee against resistance there was usually extension of the trunk and of the other three limbs with general increase of muscular contraction. Sometimes with voluntary flexion of the left knee there was flexion of the right knee and of the right elbow.

In association with voluntary extension of the right (paralysed) knee, there occurred extension of the trunk and of the other three limbs, the reaction being stronger in the right than in the left upper limb.

Speaking generally, the reaction in the remote limbs was stronger when the voluntary effort was being made in one of the lower limbs than when one of the upper limbs was the seat of the voluntary action.

It was also noticeable that the reaction of the fellow limb was more strongly developed when the voluntary effort was directed to a movement of a lower limb than to one of an upper limb.

The outstanding feature of all these associated reactions was the tonic contraction of antagonistic pairs of muscles involving in the first place the musculature of the trunk and proximal segments of the limbs, and spreading, as the voluntary effort increased, to more distant segments.

The contraction of some muscles exceeded that of their antagonists, with the result that segments of limbs were displaced. This displacement was slow, deliberate, and rarely excessive: its direction at any given joint did not usually alter throughout a reaction. This rule had some exceptions, as for example in the alternating movements sometimes observed at the wrist.

Finally, the reactions were less easily evoked in the healthy than in the paralysed limbs, and when present were usually confined to the proximal muscles and unassociated with displacement. Nevertheless, it was clear that though differing in intensity, the reaction on each side was of the same order.

Chapter IV.—Reflex Movements of the Upper and Lower Limbs in Quadriplegia and Hemiplegia.

(*A*) *Appropriate Stimuli.*

One of the reasons that led Sherrington to apply the term nociceptive to certain reflex reactions, such as the flexion reflex, in decerebrate and spinal animal preparations, was that they are constantly and most easily excited by potentially painful and harmful stimuli. Any form of nocuous stimulus is suitable, but the degree of noxa required varies under different conditions; sometimes protective reactions may be evoked by stimuli which in the normal individual would produce sensations of mild discomfort only. Thus after complete transection of the spinal cord in man vigorous flexion of the lower extremity may follow that form of light stimulation of the sole of the foot which is usually associated with a sensation of tickling ([6] p. 305). But as a rule such stimuli are effective when applied within the focus of the receptive field only and after a period of excitation of several seconds. In addition the isolated portion of the spinal cord must have reached a high degree of reflex excitability, and have become, as it were, habituated to flexor reactions by frequently recurring involuntary flexor spasms.

Under ordinary clinical conditions, however, it is comparatively rare to find an injured spinal cord that is so exquisitely responsive to stimuli: more often appropriate stimuli for the flexion reflex of the lower limb are of the painful order.

To elicit nociceptive reactions of the upper or lower limbs in our cases of hemiplegia and quadriplegia potentially painful stimuli were usually necessary. Pricking or scratching the skin with a sharp pin or pinching the tissues were usually employed. Hot or cold objects

were also effective provided their temperature was high or low enough to be capable of exciting a sensation of pain when the test object was applied to a normal part of the body. Thus No. 1 found that when he thoughtlessly grasped a cup of very hot tea with his hand, his arm went into a violent extensor spasm and the cup was flung to the ground.

The mere fact, however, that a stimulus is potentially harmful is not in itself sufficient to ensure a response. In addition to a nocuous quality a stimulus, to be effective, must possess a certain intensity and duration. We found that for nociceptive reflexes of the upper limb in hemiplegia and in quadriplegia the threshold value of stimulus was usually lowest in the palm and axilla respectively. But here a single prick stimulus usually failed to evoke a response and often a series of pricks on one spot were unsuccessful. The most effective method of stimulation was by dragging the point of a pin along the skin, or by pinching the tissues. With moderately firm pressure a response usually appeared after the pin point had travelled about one inch and the distance required was less if the pressure on the pin was sufficiently heavy to break the surface. Hence at any one point within the receptive field summation of repeated pin-pricks was more capable of exciting a response than a single deep prick. But still more effective was serial stimulation of a number of points, as in dragging the point of the pin across the skin. The effectiveness of a stimulus depended therefore as much on the extent of the surface to which it was applied as on duration and intensity. This probably explains the effectiveness of a crushing stimulus such as a pinch or squeeze of the skin and deep tissues or firm pressure with a broad object such as the end of a pencil.

The direction of the movement evoked in the lower limb can often be altered in quadriplegia when the intensity of the stimulus applied to the thigh is changed. Thus, extension of the lower limb is evoked by drawing the tips of the fingers firmly along the skin on the inner aspect of the thigh, but the movement changes to flexion when the skin is firmly pinched. We have failed, however, to obtain reversal of direction of the reflex movement evoked by increasing the intensity of the stimulus, when it is applied to either the peripheral portion of lower limb in quadriplegia or hemiplegia or to any part of the upper limb in the latter. This was the case also in the upper limb in two examples of quadriplegia, but in No. 2 strong stimulation of the palm of the hand or inner part of the forearm by pinching usually excited a biphasic reaction in the upper limb, of which flexion at the elbow and

abduction at the shoulder formed the primary movement, and extension, adduction and internal rotation the secondary movement: stimuli of moderate strength applied to the hand or forearm evoked as a rule the extension reaction only.

In this patient and in No. 3 we did not succeed in evoking a flexion reflex of the upper limb by stimulation above the elbow, but a response of this kind was usually obtained in No. 4. In No. 1 also it could be evoked by the employment of a special form of stimulus. Thus, when the strongly tonic internal rotators of the shoulder were suddenly "stretched" by the observer, a movement consisting of flexion at the elbow and wrist, flexion and abduction of the fingers and extension and abduction of the thumb was evoked. It was always quickly replaced by extension, adduction and internal rotation of the limb.

(*B*) *Reflex Movements and Receptive Fields.*

Within recent years considerable attention has been directed to the study of reflex reactions evoked by potentially harmful stimulation of the lower extremities in animals and man after lesions of the central nervous system. Work at the bedside and in the laboratory has been inspired by the researches of Sherrington, who first described and analysed in detail the flexion reflex of the lower limb in decerebrate and spinal mammals. This phenomenon follows the application of a nocuous stimulus to the foot, and consists of flexion at hip and knee and dorsiflexion of the foot with reciprocal relaxation of the antagonistic extensors [10].

Several observers have investigated the reactions of the lower limb evoked by stimulating the foot under similar conditions in hemiplegia and paraplegia, and have found that they present in general the same characters as Sherrington's flexion reflex [6] and [16]. With lesions of the pyramidal tract at any level, pricking and scratching the sole of the foot evoke contraction of the flexors of the limb including the dorsiflexors of the foot and toes. As in animal preparations reciprocal relaxation of the extensors of the limb forms part of the response.

As a rule in hemiplegia and quadriplegia, when the resting posture of the lower limbs is one of rigid extension at all joints due to excessive tone of the extensor muscles, reflex flexion can be evoked by appropriate stimulation of any part of the limb below the knee. Although the response is most readily obtained, and with stimuli of least inten-

sity, from the outer part of the sole of the foot, yet by scratching or squeezing the tissues of the leg the reaction can usually be excited. The Schaefer, Oppenheim and Gordon toe-phenomena do not essentially differ from the reaction described by Babinski [1]. Each of these observers merely recommends stimulation of different parts of the receptive field for the flexion reflex, of which "extension" of the toes forms an integral part, or the employment of different kinds of nocuous stimuli.

In the disorders we are considering, therefore, the receptive field or reflexogenous zone for the flexion reflex has its focus in the sole of the foot, but also includes the whole of the peripheral portion of the limb below the knee. Above this level it is usual to obtain the opposite movement, namely, extension of the lower limb, by harmful stimulation of moderate intensity, such as pinching, pricking or scratching the tissues of the thigh. The extension reflex response consists of contraction of the extensors of the hip and knee and the muscles of the calf and sole of the foot. Along with the contraction of the extensors, the tone of the flexors of the knee also appears to increase if the reaction is evoked when the limb is extended. The receptive field has its focus in the groin, genital and perineal regions, but includes the thigh and extends sometimes for a short distance on to the abdominal wall, especially in the middle line.

Thus the hemiplegic or quadriplegic patient with spasticity of the lower limb in extension frequently exhibits two forms of reflex reaction to harmful stimulation :—

(1) Reflex flexion at all joints, including upward movement of the toes, most readily evoked from the sole of the foot, but also obtained by strong stimulation of the foot and leg below the knee and sometimes of the thigh ; and

(2) Reflex extension of the limb, including downward movement of the foot and toes, excited by the application of appropriate stimuli to the proximal segment of the limb, especially to the parts lying near the perineum and external genitalia.

So far we have been dealing with phenomena which have already been described by Walshe [16], Riddoch [6] and others. Turning now to nociceptive reflex reactions of the upper extremity in severe spastic hemiplegia and quadriplegia, we have been able to evoke two main kinds of movements which displace the limb in opposite directions ; in one reaction the limb is extended at the elbow and in the other it is flexed. As in the lower extremity, a response readily followed appropriate stimu-

lation within a large receptive field which extended from the peripheral portion to the base of the limb and even on to the trunk. But whereas in the lower extremity excitation of the leg and foot evoked reflex flexion, and of the thigh and neighbouring portion of the trunk usually reflex extension, in the upper limb in any one patient it was more usual to obtain one form of movement only, whether the exciting stimulus was applied above or below the elbow. In some of the cases, chiefly hemiplegic, flexion at the elbow, and in others, chiefly quadriplegic, extension at this joint occurred, but the receptive fields for both reactions were approximately the same.

In one quadriplegic patient (No. 2), however, either flexion of the elbow with abduction of the shoulder or extension of the elbow with adduction of the shoulder could be evoked by stimulation of the receptive field below the elbow; but appropriate stimulation above the elbow evoked an extension reaction only.

We propose to describe the first form of response as an "extension reflex of the upper limb." It was the response which constantly formed the fully developed nociceptive reflex reaction in three of our four quadriplegic patients, and in one patient with hemiplegia. It is well illustrated in the serial kinematograph pictures on p. 412. It is a vigorous movement which thrusts the extended upper limb backwards, at the same time producing extreme inward rotation, so that the palm of the hand at the height of the response faces outwards as well as backwards. The movement, when analysed at the different joints, is found to consist of elevation, adduction, retraction and inward rotation at the shoulder, extension at the elbow, hyperpronation of the forearm, flexion at the wrist and extreme adduction and extension of the fingers and thumb.

The movement is so rapid and strong that it was found to be impossible to determine by inspection and palpation the state of the antagonists while it was taking place—although they were certainly contracted while the position produced by the reflex was being maintained.

The receptive field for the reflex includes the palm of the hand and the palmar aspect of the fingers and thumb, the flexor and inner aspect of the forearm and upper arm, the axilla and the upper part of the chest, in front and behind, from about the second to the sixth ribs. The back of the hand and fingers and the posterior and outer aspects of the limb were usually unresponsive, and a fully developed reaction could not be excited from these parts. The most excitable portions of

the field were the four inner walls of the axilla and the palm of the hand, and the former more than the latter. That the axilla and inner aspect of the upper arm form the focus or part of the receptive field, where the threshold value of stimulus is lowest, is supported by the following observations:—

(1) It seems to be the part of the limb which is the earliest to respond to stimulation after the lesion of the spinal cord. For evidence on this point we are dependent upon observations in one case only made in No. 1 by Lieut.-Colonel Gordon Holmes five days after the injury. He noted that with the application of several pin-pricks in series to any part of the upper arm from the internal epicondyle to the axilla, a strong reflex movement occurred, consisting of adduction and inward rotation of the arm at the shoulder. By the time the patient came under our care, thirty-five and a half months after the injury, the receptive field had spread to its full extent.

(2) In all the quadriplegic patients who showed this reaction it could usually be evoked more easily from the axillary region than from the palm throughout the period of observation, which in No. 1 extended over six years. The difference in the thresholds for palm and axilla, however, was not great in any of our patients at a stage when their condition had become stationary.

(3) The minimal motor response from any part of the receptive field consisted most frequently of the movement which was the earliest reaction to appear after the initial spinal shock had passed away. In two of the patients the first movement to be evoked by stimulation of the upper arm or the chest was adduction and inward rotation at the shoulder, while in No. 3 the minimal response was extension of the elbow and flexion of the wrist and extension and adduction of the fingers.

The receptive field was strictly homolateral in all our examples of hemiplegia and quadriplegia, with one exception in which it was bilateral for the reflex in one upper limb. This patient (No. 3) was paralysed in all four limbs following a shell wound of the neck, but within two hours of his injury his right upper and lower extremities began to recover. By the time he came under our observation three days later all voluntary movements of his limbs on the right side could be performed, although his left upper and lower extremities were helpless except for feeble voluntary contraction of deltoid and biceps. Within a few months his right upper and lower extremities had, as regards motion, almost completely recovered. Analgesia of the inner

aspect of the upper limb however persisted. Return of voluntary power in his left upper extremity was slow, and the limb showed rigidity of the same kind and distribution as in Nos. 1 and 2. Reflex extension was obtained in his left upper limb only, but the receptive field was bilateral and practically symmetrical, comprising the palms of the hands, the inner aspect of both limbs and the upper part of the chest in front and behind from about the second to the fifth ribs.

The second form of nociceptive reflex reaction of the upper limb to which we draw attention in this paper was obtained in hemiplegic patients, and in two examples of quadriplegia (Nos. 2 and 4). Paralysis was, as in all our cases with one exception (No. 8), associated with spasticity, but in this group the rigidity preponderated in the flexors over the extensors of the upper limb.

The reflex response, for which a convenient term is "flexion reflex of the upper limb," was readily obtained in No. 5. On scratching the palm of the paralysed hand there occurred flexion of fingers, wrist and elbow, slight abduction and external rotation of the upper arm and elevation of the shoulder. These were the main components of the general response which varied in details according to the situation of the stimulus within the receptive field. This was invariably homolateral and corresponded almost exactly in extent with the receptive field for the extension reflex, covering the palmar aspect of the hand and fingers, the flexor and inner aspects of the forearm and upper arm, and spreading over the axilla and upper part of the chest on the same side. The focus of the receptive field, however, was differently situated in the two groups; it was peripheral in the palm for the flexor reaction in hemiplegia, and proximal in the axilla and upper arm for the extension reflex in quadriplegia. In the one example of quadriplegia (No. 4), however, in which flexion at the elbow was the most common response obtained at this joint, it was most easily elicited, as in the other cases of quadriplegia, from the axillary region.

Although flexion at the elbow in this patient usually formed part of the general reaction of the limb the response at other joints was different from that obtained in hemiplegia. For example, the movement evoked by scratching the palm or axilla consisted of adduction, elevation and outward rotation at the shoulder, flexion at the elbow and extension of the fingers and hand. This patient showed remarkable variability of response with alteration in the locality of the exciting stimulus, and the spasticity and paralysis of the limb were relatively slight; but a patient with a similar form of quadriplegia, who has just come under our observation, shows a reaction of the same kind.

In another example of quadriplegia (No. 2) with extensor rigidity of the upper limbs, flexion of the elbow with abduction and elevation of the shoulder could sometimes be evoked by strong stimulation of the tissues on the ulnar aspect of the forearm and the palm of the hand. The response was a quick unsustained movement which was followed by the more usual extension response. It was more readily evoked as a bilateral reaction by simultaneous stimulation of both upper limbs below the elbows.

All the hemiplegic patients examined did not exhibit the fully developed response. In many the reaction was limited to slight flexion of the fingers and hand with perhaps some pronation of the forearm, and in these the receptive field was confined to the palm. Thus the minimal motor response to harmful stimulation in hemiplegia with spasticity is often flexion of the fingers, and the focus of the receptive field the flexor aspect of the palm and digits. On the other hand in No. 7 we were able to observe the development of this reflex while the right upper limb remained completely paralysed and flaccid. The first movements to appear were abduction and external rotation at the shoulder accompanied at a later stage of recovery by flexion and slight supination at the elbow.

(*C*) *Influence of the Locality of the Stimulus on the Movements evoked—Local Sign.*

From the fact that one and the same nociceptive reflex in either the upper or the lower limb can be excited by stimulation of widely separate points within a large receptive field, it might have been assumed that considerable variations in the reactions evoked would be obtained. This, however, was not found to be the case except when the paralysis was slight as in No. 4. Although minor modifications were evident, depending mainly on the relative amount and duration of contraction in those muscles which were in most intimate nervous connection with the region stimulated, the general form of the response of the limb as a whole remained true to type. For example, when reflex flexion of the lower extremity was evoked by stimulating the outer part of the sole, with dorsiflexion of the foot there tended to be eversion, which was replaced by inversion and adduction when the locus of the stimulus was on the inner border of the foot. Again, in the reaction obtained by pinching the tissues round the knee, flexion at the knee and hip was sometimes relatively

greater than the movement of dorsiflexion of the foot and toes. But whichever part of the limb was stimulated within the receptive field for the reflex, flexion at all joints was the basic element of the response.

In the upper limb also, in cases of severe hemiplegia and quadriplegia, the nociceptive reactions evoked from the axilla and the palm of the hand were almost identical in general form. Variations attributable to the locality of the stimulus when present appeared in the initial part of the response before the general reaction developed completely. For example, pronation of the forearm and flexion of the wrist and fingers were early movements to follow stimulation of the ulnar part of the hand in quadriplegia, while extension and adduction of the fingers and thumb were usually evoked more easily from the thenar eminence. So also extension of the elbow and adduction and inward rotation of the shoulder were prominent when the stimulus was applied near the elbow and shoulder respectively. But as soon as the full reflex effect developed these differences disappeared, the complete reaction being the same from whichever part of the receptive field it was evoked.

Examples of severe hemiplegia showed in the nociceptive reaction in the upper limb a similar relative independence of the locus of the stimulus.

The reflexes we have been considering in the upper and lower limbs in hemiplegia and quadriplegia, therefore, belong to types. In each case appropriate stimulation of a large reflexogenous zone, which possesses a focus or central area where the threshold value of stimulus is lowest and a fairly well defined boundary, evokes a response which in form and direction of displacement of the corresponding limb-segments is almost but not absolutely stereotyped. With minimal reactions definite variations in the motor effects excited can be traced to the locality of the stimulus ; but as soon as the complete or maximum reaction is evoked modifications become submerged and disappear.

Reduction in the adaptability of the reflex response to the site of nocuous stimuli has already been described in cases of paraplegia in flexion following transection of the spinal cord in the thoracic region. In these cases the prominent feature of the reflex picture is a massive reaction consisting of a bilateral flexor spasm of the trunk and lower extremities with evacuation of the bladder and rectum and excessive sweating. The receptive field comprises the whole of the paralysed region of the body and the situation of the stimulus has little effect on the form or extent of the movement except in sub-total reactions ([6] p. 345 and [4] p. 217).

There is one case in our series, however, in which reactions in the upper limb were influenced considerably by the situation of the exciting stimulus. The case No. 4 has already been touched upon in other connections and may profitably be referred to again in so far as it throws light on the present problem, but we hope to deal with it more fully in a later communication.

The patient at the age of 17 years suffered from an illness which was diagnosed as influenza. When he was allowed to get up after three weeks in bed, he noticed that his lower limbs were weak. The disorder slowly increased and within a year after the onset his left upper limb became affected in the same way. Later his right upper limb showed signs of impaired motor function. At the present time, three years after the attack of influenza, he shows moderate weakness and spasticity of all four limbs. He suffers little pain, and there is slight disturbance only of postural sensibility in his toes and of vesical and rectal functions. The functions of his cranial nerves are normal and radiographic examination of his spine fails to reveal any abnormality.

Muscular tone is definitely increased in his limbs and trunk, more on the left than on the right side, but not to any marked extent. In his upper limbs the excess of tone is about equal in all muscle-groups at the shoulders, at the elbows and wrists the flexors are more tonic than the extensors, but the fingers are hypotonic. In his lower limbs the extensors perhaps offer more resistance to passive movements than do the flexors.

From a large receptive field, comprising the whole upper limb the shoulder and the upper part of the chest, nociceptive reflex movements can be evoked. These reactions are most easily obtained with weak stimuli from the axillary walls and the inner aspect of the upper arm; for in this region a gentle moving contact on the skin with the tip of the finger is capable of evoking a movement of the limb. The back of the hand and the outer aspect of the forearm and upper arm are much less responsive.

The movement most commonly obtained consists of adduction and external rotation at the shoulder, flexion at the elbow and extension of the hand and fingers, but the response varies in a remarkable manner with alteration in the locality of the stimulus. Thus when the reaction is excited by scratching the palm of the hand, the movement at the shoulder is abduction and retraction of the upper arm. Stimulation of the back of the forearm yields adduction at the shoulder and of the

inner aspect abduction at this joint. Again, when the stimulus is applied to the skin over the deltoid, the response is mainly strong elevation of the shoulder with adduction and external rotation of the upper arm.

Without enumerating, for the present, further examples of reactions evoked in this patient, we wish to make clear that variability of response with change in the situation of the stimulus is an outstanding feature in this case, in which disturbances of voluntary power and muscular tone are relatively slight.

A comparison of the reactions in this patient with those in more severe cases of quadriplegia and hemiplegia justifies the conclusion that invariability of the response and enlargement of the receptive field for any one protective reflex are indicative of the extent to which disintegration of neural function has followed a lesion of the central nervous system; from this point of view they have the same significance as spasticity and paralysis.

The larger the field of a protective reflex the less do the reactions vary with the locality of the stimulus and the more restricted is the reflex behaviour of the limb as a whole.

(*D*) *Motor Phenomena accompanying the Reflex Movements of the Upper and Lower Limbs.*

Three of the quadriplegic patients (Nos. 1, 2 and 3) showed extensive reflex reactions involving the trunk and limbs.

Irradiation of reflex effect beyond the limb directly stimulated was very striking in Nos. 1 and 2, and was obtained with either reflex flexion of the lower extremity or reflex extension of the upper limb when the response was powerful. For example, in No. 2, the following reaction was evoked by applying a strong nocuous stimulus within the receptive field on the right upper extremity. The stimulated limb was thrust violently backward with adduction and inward rotation of the upper arm, strong extension and hyperpronation of the forearm, so that the palm of the hand faced outwards, flexion of the hand and extension and adduction of the fingers. On the left side all the muscles of the upper limb above the wrist were brought into action, but some more strongly than others, so that some displacement of the different parts of the limb occurred; there was slight adduction of the upper arm with forward movement of the shoulder, flexion and supination of the forearm and slight extension and radial deflection of the hand.

Palpation showed that erector spinæ and the anterior abdominal muscles contracted on both sides, and there was some flexion and rotation of the trunk to the right. Both extensors and flexors of the lower extremities became strongly contracted, the former more than the latter, so that there was rigid extension at hips, knees, and downward movement of the feet and toes; the right lower limb was more affected than the left.

The whole response obtained by stimulation of the palm of either hand in No. 1 was practically identical with that just described. There was augmentation of tonus in both prime movers and antagonists of the trunk, lower extremities and the crossed upper limb, and similar slight displacements at the different joints were observed. The only noteworthy difference was that the rotation of the trunk to the stimulated side was more prominent, and was accompanied by slight flexion at the hips when the patient was standing erect.

From this fairly uniform response, however, variations of some note occurred in the case of No. 3. It has already been mentioned that this patient was wounded by a piece of shell in the cervical portion of the spinal cord on July 15, 1916, and was for some time paralysed in all four limbs; but by March, 1917, his right upper and lower extremities had almost completely recovered. At this time the nociceptive extensor reaction was obtained in his left upper limb only. The reflex could, however, be excited by appropriate stimulation, not only from the palm and the inner half of the upper limb and the upper part of the chest on the left side, but also from the corresponding parts on the opposite side. Thus on scratching the skin of the right forearm, while no apparent change occurred in the muscles of the right arm, a typical extension response was evoked in the crossed upper limb. In this patient, then, there was identity in the forms of response excited by stimulating corresponding parts within the receptive field on the same and opposite sides. The motor effects produced by the activity of the reflex spread to the left lower extremity, and the direction of displacement noted on March 27, 1917, is another point of divergence from the reactions in Nos. 1 and 2. Whereas in the latter the remote results in the lower extremities of nocuous stimulation of an upper limb was invariably bilateral extension, in No. 3 they were unilateral and consisted of a flexor spasm of the left limb.

Scratching or pinching the toes or foot so as to excite strong reflex flexion of the lower extremity usually yielded results which were equally extensive. For example, in No. 2 a vigorous flexor reaction in

the left lower extremity was accompanied by the following changes in the musculature of the other three limbs and the trunk: Extension at all joints of the right lower extremity including downward movement of the foot and toes, but tonic contraction of the flexors (hamstrings and pretibial muscles) also occurred; vigorous contraction of the erectores spinæ and recti abdominales, the former more than the latter, and more on the stimulated side; in the left upper limb, slight but definite increase of tone in the adductors and abductors of the shoulder and in biceps and triceps with extension of the elbow; in the right upper limb, contraction of the adductors and abductors of the shoulder, biceps and triceps with adduction of the upper arm and slight flexion of the forearm.

A reaction of the same kind followed excitation of reflex flexion of the right lower limb. In this case triceps contracted more strongly than biceps in the homolateral upper limb.

Stimulation of the flexion reflex in the left lower extremity in No. 3 evoked as in No. 1 extension of the crossed lower limb, flexion at the elbow and adduction at the shoulder in the left (homolateral) upper limb and extension at the elbow, wrist and metacarpo-phalangeal joints in the right (crossed) upper limb.

In No. 1 the response in the crossed lower extremity and in the trunk was the same as in No. 2, but in the arms a stronger and more widespread reaction was obtained. Sometimes movements of all segments of the upper limbs were observed, and usually consisted of adduction of the upper arms, flexion and supination of the forearms, extension of the hands and of the fingers at the metacarpo-phalangeal joints with flexion at the interphalangeal joints. These displacements, however, were never extensive, especially at the proximal joints; and invariably palpation and inspection showed that, as in the associated reactions in No. 2, the antagonists of the muscles which brought about the displacements at the different joints did not become relaxed, as happened in the flexion reflex, but, on the contrary, were more contracted than before.

Summarizing our findings in three examples of quadriplegia, regarding change of attitude in the limbs and trunk effected by nocuous stimulation of a hand or foot, the following general conclusions are arrived at:—

(1) Stimulation of the hand or forearm evoked in Nos. 1 and 2 an extension response in the limb and adduction of the upper arm and flexion of the forearm in the crossed upper limb. To this rule

No 3 is an exception. In this case stimulation of the left hand or forearm gave the extension response which was not accompanied by evident changes in the crossed upper limb. Also stimulation of the right forearm evoked a reaction in the left upper limb only and at the elbow extension and not flexion was observed.

(2) The response in the lower limbs which followed appropriate stimulation of either upper limb in Nos. 1 and 2 was always bilateral extension at knee and ankle. In No. 3 only one experiment was made to investigate this point and in it a flexor spasm of the left lower limb accompanied an extension response in the left upper limb.

(3) Nocuous stimulation of the foot invariably evoked reflex flexion of the stimulated limb and extension of the crossed lower limb. In No. 2 the crossed response consisted of extension at hip and knee and downward movement of the foot and toes, while in No. 3 dorsiflexion of the foot and toes was sometimes observed.

(4) The remote effects in the upper limbs from excitation of reflex flexion in one lower limb were variable. In Nos. 2 and 3 the usual result obtained at the elbow was extension in the crossed limb and flexion in the homolateral limb. In No. 1 bilateral slight flexion was the rule.

Similar reactions were observed in our hemiplegic patients, but the response on the healthy side was comparatively feeble, especially in the upper limbs.

Sherrington has described the postural alterations in the limbs and trunk, which, in the decerebrate and high spinal animal preparations, follow nocuous stimulation of the fore and hind paws ([8] p. 165). They show a certain variability as regards the direction of displacement of the limbs in different preparations and in different experiments with the same preparation, but on the whole there tends to be a diagonal correspondence between fore and hind limbs. That is to say flexion of one fore limb is accompanied by flexion of the opposite hind limb and extension of the other two.

The order in which the different parts were involved in the reaction seemed to be the same in our patients as that described by Sherrington for animals. For example, stimulation of the right hand evoked movement of (1) the stimulated limb, (2) the trunk, (3) the right lower limb, (4) the left lower limb, and (5) the left upper limb. Sometimes, however, the crossed upper limb appeared to be innervated earlier than the lower limbs, but usually it seemed to be brought into action later and generally it was affected less than the rest of the body. As

regards the lower limbs the homolateral showed a stronger reaction than the contralateral.

With excitation of the flexion reflex of the lower extremity the sequence of involvement in the resulting reaction was: (1) the stimulated limb; (2) the opposite lower limb; (3) the trunk; (4) the homolateral upper limb; and (5) the crossed upper limb.

It was noticeable that stimulation of one lower extremity led to early and pronounced effects in the other, whereas in the case of the upper limbs the crossed reaction as a rule was relatively slight. Thus it would seem that the lower limbs are, as regards reflex connections, coupled together more closely than are the upper limbs. The former tend to react to reflex stimulation as a unit, while the latter are more independent of one another.

The sequence of irradiation, or order of spread, of reflex effect within each limb taking part in the associated reaction followed a definite rule which never varied. In the development of a complete reaction in association with a vigorous nociceptive reflex response of an upper or lower limb the "march" of innervation in each of the other three limbs was from base to periphery. Thus the muscles of the shoulder or hip contracted before a definite change could be noticed in those of the elbow or knee and these again before the muscles of the wrist or ankle. The field of musculature most affected outside the limb directly stimulated comprised the trunk and the proximal segments of the limbs. Increase in the intensity of the reaction led to: (1) augmentation of muscular contraction in these parts; and (2) progressive involvement of muscles more peripherally placed in the extremities.

It has already been pointed out that any displacement of the trunk and three limbs that occurred in the associated reaction was rarely extensive, although the muscles were frequently in strong contraction. This was undoubtedly due to the innervation of antagonistic muscles, which also explains another distinctive feature of the associated movements, namely, their slowness. The movements of the limb and trunk, in comparison with the rapid displacement of the stimulated limb, were carried out slowly, almost "deliberately." Each was a steady smooth performance, a gradual alteration of attitude rather than a movement. It was also generally uniphasic in the sense that for each segment the direction of displacement did not change in the reaction even when alternating movements of the stimulated limb were evoked by a prolonged stimulus. For example, by applying a continuous strong pinching stimulus to a toe, flexion of the lower limb was not usually main-

tained at a steady level, but was repeatedly broken through momentarily by active extension of the limb. The biphasic response thus evoked, however, was in general limited to the lower extremity under stimulation, the rest of the body maintaining a uniform posture.

(*E*) *Reflex Movements in Relation to Tonic Postures of the Limb at rest.*

Although almost all the muscles of the affected limbs were spastic, yet some were more hypertonic than their antagonists and so determined the postures of the limbs at rest. Thus in hemiplegia the upper limb is held in a position of flexion and the lower limb is extended. Extension was also the resting attitude of the lower limbs in the four examples of quadriplegia recorded in this paper, but while in one the upper limbs were slightly flexed, in the other three they were as a rule strongly extended at the elbows.

Extensor rigidity of all four limbs is the attitude usually observed in decerebrate mammalian preparations [12] and it may be helpful, for purposes of comparison with our own clinical observations, to summarize Sherrington's records of nociceptive movements in anthropoids in relation to the postural rigidity of the limbs ([9] p. 412).

Sherrington found that there is mutual antagonism between the results of nociceptive stimulation and the reflex posture of the limbs at rest, not only in the sense that the former are concerned with movements and the latter with maintained attitudes, but in the fact that muscles which are opposed in function are employed. Thus when the pad of the hind foot is pricked a movement of flexion of the limb is evoked and with contraction of the flexors of the knee and ankle there is simultaneous relaxation of the previously tonic extensors. A similar result follows harmful stimulation of the fore paw; when it is dipped into hot water flexion is evoked at the elbow, which at rest is tonically extended. He obtained results of the same kind in the decerebrate dog and cat. Nociceptive reflexes excited by stimulation of the peripheral portions of the limbs thus produce flexion at knee or elbow with reciprocal inhibition of tone in the corresponding extensors.

Innervation in the same limb by nociceptive reflexes of muscles which are opposed in function to those employed in postural reflexes, is however not a universal rule. There is at least one exception in the lower limb. In the decerebrate cat, dog and monkey, reflex flexion is the result which usually follows harmful stimulation of the greater part of the hind limb, but when the stimulus is applied to the thigh near the groin or to the perineum, extension and not flexion is evoked. Thus

the nociceptive reflex, which is excited from the base of the hind limb, employs the same muscles as are in tonic contraction when the limb is at rest.

So far as we know a nociceptive extension reflex has as yet not been described for the upper limb in laboratory preparations.

Let us now examine our clinical observations in the light of Sherrington's results in decerebrate animals. The postural attitude of the lower limbs in both hemiplegia and quadriplegia was extensor, due to preponderating tonicity of the extensor over the flexor muscles. This rigidity was inhibited and extension replaced by flexion when appropriate harmful stimuli were applied to the foot or to the leg below the knee. Sometimes reflex flexion could be obtained from the thigh by using intense stimuli. Usually, however, with stimuli of moderate strength extension and not flexion of the limb was evoked from the thigh, especially near the groin. Thus, in respect of the lower limb, the postural and nociceptive reactions correspond to those described by Sherrington in decerebrate mammals. Opposite effects result from harmful stimulation of the peripheral and basal portions of the limb. From the foot reflex flexion is evoked with inhibition of the tonic extensor rigidity and from the groin reflex extension. In the former case the nociceptive reflex employs the antagonists of the muscles which maintain the resting attitude of the limb, while in the latter the same muscle groups are innervated by the nociceptive and postural reflexes.

On making a similar analysis of the postural and nociceptive reactions of the upper limb, it is evident in the first place that, unlike the case of the lower limb, a single uniform resting attitude does not hold. Examining, for example, the posture at the elbow-joint in three examples of quadriplegia, there was as a rule bilateral tonic rigidity of triceps with maintained extension of this joint when the limbs were at rest. Occasionally, in one example (No. 2), after the limb had been kept flexed for some time either passively by the observer, or through the voluntary efforts of the patient, tonus was found to predominate in biceps. But this was not the usual state. In No. 4 spasticity was less developed and more equal in antagonistic muscle-groups, but it was always greater in the flexor at the elbow than in the extensor. A resting posture of flexion at the elbow is well recognized in spastic hemiplegia and is almost the invariable rule. The occurrence of one posture rather than another in these states is of course due to an unequal development of tonus in antagonistic muscle-pairs; in severe quadri-

plegia with well developed spasticity, the tonicity of triceps is greatly in excess over that of biceps, and in hemiplegia with rigidity the opposite state obtains. As regards the posture at the other joints of the limb in the two groups of cases there is less difference. In general the upper arm is adducted and rotated inwards, the forearm pronated and the wrist and fingers flexed.

In quadriplegia, with the exceptions of No. 4 and, under certain circumstances, No. 2, whose cases will be considered separately, the nociceptive reflex of the upper limb when excited generally brought into action those muscles which by their extensive tonic contraction determined the resting posture of the limb. For example, in Nos. 1, 2 and 3, the upper limb when at rest was held in the following position : Adduction and inward rotation at the shoulder, extension at the elbow, pronation of the forearm, and flexion at the wrist. Potentially harmful stimulation of the palm evoked a powerful movement of the limb, in which the upper arm was adducted and rotated inwards, the forearm extended and hyperpronated and the hand flexed. It must be again pointed out, however, that in No. 2 strong stimulation of the forearm or hand sometimes evoked a movement of abduction at the shoulder and flexion of the elbow ; but it was quickly replaced by adduction and extension.

In hemiplegia, it is not the rule to find all the same muscles employed in nociceptive reflexes as in the postural reaction at rest. For example, the usual postural reaction at the shoulder is one of internal rotation and adduction, whereas the movement produced by nocuous stimulation is the result of contraction by the external rotators and abductors. Although flexion of the elbow, wrist and fingers is the general rule in both reflex reactions the movement of the forearm in the nociceptive reflex is always slight and in some cases tends to supination rather than pronation, the usual postural attitude of this segment of the limb at rest.

We have been comparing nociceptive reactions with postural reactions in cases of severe hemiplegia and quadriplegia in which spasticity and paralysis were well marked. One example of quadriplegia (No. 4) with much less spasticity and paralysis presented features of importance in relation to those which have just been discussed. In this patient the disturbance of function due probably to a high cervical lesion was altogether less than in the other cases of quadriplegia. All voluntary movements were fairly strong and free ; sensation, apart from slight loss of postural sensibility in the feet and toes, was unaffected, and the vesical defect amounted to slight frequency of

micturition only; there were increased tendon-reflexes in the four limbs and the abnormal form of plantar response was present on both sides, but spasticity was relatively slight and was more evident in the lower than the upper limbs. Tonus however was quite definitely increased in all the affected muscles, but although it was, as in our other cases, unequally developed in antagonistic muscle-groups the inequality was not great. In the trunk and lower limbs, the extensors were somewhat more spastic than the flexors and there was difficulty in clearing the ground with the toes in walking. In the upper limbs the flexors of the wrists and pronators of the forearms resisted passive movements more than their antagonists and in this respect there was no difference from the distribution of tonus in the other examples of quadriplegia. But at the elbows the flexors and not the extensors were the more spastic muscles, and at the shoulders tone did not predominate in any one muscle group over another.

Nociceptive reflex reactions were easily excited in either upper limb especially from the axilla and inner aspect of the upper arm where a gentle moving contact with the tip of the finger was an effective stimulus. The reactions showed great variability with alteration of the site of the stimulus, but from the axilla the usual movement consisted of quick elevation, adduction and external rotation at the shoulder, slight flexion at the elbow and extension of the wrist and fingers. Flexion, however, was not the invariable movement at the elbow; extension usually occurred when the stimulus was applied to the posterior aspect of the forearm or upper arm near the elbow.

In this patient therefore in whom the paretic muscles were not excessively hypertonic the nociceptive reactions, which were determined by the site of the stimulus, showed no definite relationship to the distribution o muscular hypertonus or posture of the limb at rest.

Thus the correspondence between the muscles employed in the nociceptive reflex of the upper limb and those determining the resting posture is almost complete in many cases of severe quadriplegia, less intimate in cases of severe hemiplegia and practically absent in the case of slight quadriplegia we have just described.

On the other hand in all cases there were two forms of nociceptive reflex in the lower limb—one employing the muscles determining the extended posture of the limb at rest, the other bringing into action their antagonists.

These facts appear to show that nociceptive reactions are inde-

pendent of postural rigidity in the upper as compared to the lower limb, the independence being less marked the more severe the disintegration of function of the central nervous system.

(*F*) *Motor Phenomena following Primary Reflex Movements.*

Displacement of the limb in a particular direction is not the only consequence of the application of a harmful stimulus. The primary movement, after it has reached its height, is succeeded either by an active movement, which is opposite in direction to the first (rebound phenomenon of Sherrington ([8] p. 208), or by active maintenance of the attitude into which the limb has been placed by the movement, until gradual relaxation supervenes.

In cases of quadriplegia and hemiplegia with extensor rigidity of the lower limb, when the stimulus is applied below the knee, the former mode of reaction is usually observed, flexion followed by extension. This secondary movement of extension, however, is not a passive phenomenon in the sense of being due only to relaxation of the flexors and the weight of the limb. It is the consequence of active contraction of the extensors of the hip, knee and ankle and of the short muscles of the sole of the foot. Generally speaking, the strength of the secondary extensor contraction varies directly with (1) the strength of the primary flexion of the limb and (2) the degree of extensor rigidity present in the limb at rest.

Sherrington's analysis of similar biphasic movements of the hind limb in the decerebrate cat shows that during the execution of the primary movement of flexion the extensors are reciprocally inhibited. When the movement of flexion has reached its height and the flexors begin to relax, the extensors are innervated and the resulting contraction, which extends the limb, is stronger than the tonic contraction of these muscles before the reflex was excited (post-inhibitory exaltation). If with the limb at rest the degree of tonic contraction in the flexors is approximately equal to that in the extensors, as in the spinal cat or dog, then the secondary extension may be followed by flexion and this again by extension (reflex steppage movements).

This phenomenon of serial alternating reflex movements of flexion and extension can sometimes be obtained in cases of paraplegia ([7] p. 49) and quadriplegia in man when the degree of extensor tonus approximates that of flexor tonus in the lower limb (No. 4). But when hypertonus preponderates in the extensor muscles, as in hemiplegia and

most of our examples of spastic quadriplegia, then harmful stimulation of the foot evokes a single biphasic movement of flexion followed by extension, the series of alternating movements being interrupted at this point. The probable explanation of this event is that post-inhibitory exaltation of the flexor reflex arcs at the end of the extension phase of the movement is insufficient to overcome or inhibit the extensor hypertonus which becomes automatically reasserted.

In hemiplegia and quadriplegia, with extensor rigidity of the lower limb, reflex flexion evoked by the application of an appropriate stimulus is thus not maintained after the completion of the movement, but is inevitably followed by a movement of extension which brings the limb back to its usual position of rest. Although the reaction to a brief stimulus is limited to a simple biphasic movement of flexion followed by extension, a more prolonged stimulus is usually followed by a more complex response.

In this case a series of movements, extension alternating with flexion, separate the complete primary flexion of the limb from its final extension. These movements, however, are of small range and often of irregular rhythm. Although alternating movements of this kind may persist for a considerable period while steady stimulation is being maintained, they gradually die out and disappear, the limb being finally left in its previous state of tonic extension.

A reaction in which extension forms the first phase may also be evoked by stimulating the lower limb. This, it will be remembered, can be obtained by a stimulus applied to the thigh. If the stimulus is sufficiently strong, the primary movement of extension is followed by one of flexion, which again is succeeded by extension. The triphasic character of this reaction probably depends on the fact that, the initial movement being one of extension, three phases are necessary to bring the limb back to its resting attitude determined by its extensor rigidity. This may be compared to the biphasic result of stimulating the foot when the first movement is one of flexion and only a second is necessary to replace the limb in its extended position.

Active maintenance of the attitude into which a limb has been placed as the result of nocuous stimulation is the other kind of motor phenomenon to follow the primary movement. In the cases under consideration this occurs only when the primary reflex movement is one of extension of the limb and, therefore, is limited to reactions which commonly follow stimuli of moderate intensity applied above the knee. The prolonged contraction of the extensor muscles following a brief

stimulus must be regarded as an essential part of the reflex action. Relaxation takes place gradually after a few seconds and the limb is finally left with its original degree of extensor rigidity. In cases of spastic paraplegia with flexor rigidity this mode of reaction, represented by a prolonged contraction of the flexor muscles, is a common sequence of reflex flexion.

We have observed, therefore, two varieties of reactions following the application of a nocuous stimulus to the lower limb. The first is represented by alternating movements, which may be biphasic or triphasic according to whether the primary movement is one of flexion or extension. In the second there is a temporary maintenance of the posture produced by the primary movement.

As far as our experience goes nocuous stimulation of the upper limb usually fails to produce biphasic or triphasic phenomena following the primary reflex movement. According to the strength of the stimulus either a simple momentary displacement is as a rule evoked, or this new posture is maintained for a few seconds and then gradually fades away.

In hemiplegia, for instance, stimulation of the palm of the hand is followed by flexion, rarely by extension, of the elbow. In either case the after-effect of a stimulus of moderate intensity is to maintain for a time the displacement produced by the primary movement.

So also in quadriplegia, the same phenomena following the primary reflex movement are observed although the latter is more often extension than flexion at the elbow. In No. 2, however, a biphasic reaction in the upper limb could sometimes be evoked by strong stimulation of the flexor aspect of the forearm or hand. It consisted of flexion of the elbow and abduction of the shoulder quickly followed by strong extension and adduction.

The rarity in the upper limb of alternating movements such as occur in the lower limbs might at first sight be ascribed to the development of excessive tone in certain muscular groups. It is true that the muscles, which determine the direction of the reflex movements in either severe hemiplegia or quadriplegia, are generally those carrying a preponderating amount of tone when the limb is at rest. To this rule there are certain exceptions. For instance, in hemiplegia, whereas the adductors and internal rotators of the shoulder are more tonic than their antagonists at rest, abduction and external rotation are prominent features of the reflex movement. Moreover, it must be remembered that biphasic and triphasic movements can be evoked in the lower limb in spite of an

excessive extensor rigidity prevailing while the limb is at rest. Finally, we have been able to elicit a reflex movement without biphasic character in the upper limb of a hemiplegic patient, while his paralysed arm was completely flaccid and without tendon jerks.

A more probable explanation lies in the fact that the lower limb is primarily an organ for locomotion, a form of activity to which alternating movements are essential. On the other hand the inherent function of the upper limb is prehension, an action characterized by uniphasic flexion.

Chapter V.—Postural Reactions in Quadriplegia and Hemiplegia.

(*A*) *Nature of Spasticity.*

There can be no doubt that the phenomena which have been described in the preceding chapters are reflex in origin, although clearly they are not all of the same order. Judged by the effects produced it is possible to distinguish two groups, in one of which the reflex activities are expressed as postures, and in the other of which reflex movements involving displacement are manifested.

On the basis of this distinction it is our purpose now to compare some of the reflex reactions in quadriplegia and hemiplegia with those of a similar nature which have been investigated by physiologists in mammalian preparations and by clinicians in spastic paraplegia in man.

The state of maintained muscular contraction or rigidity which, under favourable conditions, follows the removal of the cerebral hemispheres in the monkey, cat, dog, and other animals, was first described by Sherrington in 1896 ([9] p. 411). Since then he has carried out extensive studies of the phenomenon, and has shown conclusively that it is reflex in origin and is postural in function—the posture which it subserves being the maintenance of the upright position of the body. In other words, it is reflex standing. In decerebrate rigidity he has described certain characteristic features the recognition of which has played an important part in the investigation of spastic states in man. Investigations along these lines have already been made upon the extensor rigidity of the lower limbs, which may follow lesions of the spinal cord ([16] p. 269 and [6] p. 329). In patients suffering from paraplegia of this form the lower extremities are rigid in extension from excessive developments of reflex tonus in the extensors of the hip, knee, and ankle (calf muscles). This rigidity has been found to

respond to tests in the same way as the rigidity of decerebrate mammalian preparations, and it appears to have the same functional significance. For when it is present the weight of the body can be supported in the upright position even in cases where voluntary power over the lower limbs is lost.

Hitherto, so far as we know, the spasticity of the somatic musculature in cases of quadriplegia has not been investigated in the light of Sherrington's researches, and the resemblance in most respects between the rigidity in our cases and that of the decerebrate animal is sufficiently striking to make the comparison a matter of some interest.

The distribution of the rigidity in the decerebrate preparation in the resting state is fairly constant, and determines the attitude of the body and its component parts. Sherrington ([9] p. 412) gives the following description of the posture of the decerebrate monkey when it is suspended in a cradle with its limbs hanging down. The fore-limbs are retracted at the shoulder-joints, straightened at the elbows and somewhat flexed at the wrists; the limbs are so rotated that the palmar surfaces of the hands face inwards. The hind-limbs are similarly retracted, extended at the hips, very stiffly at the knees, and to a less extent at the ankles. There is slight opisthotonos in the lumbo-sacral region, and the head of the animal is kept elevated with the chin tilted forwards.

The points of similarity between this attitude and the posture assumed by No. 1 in standing are in many respects very close. It is true that the slightly flexed attitude of the trunk in Nos. 1 and 2 is in contrast with the lumbar opisthotonos of the monkey, but this difference may be accounted for by the fact that the respective descriptions correspond to the erect position of the former and the horizontal prone attitude of the latter. It is in relation to the limbs, however, that the postures are most in agreement. The lower extremities are extended at all joints and the upper limbs adducted, internally rotated, but not markedly retracted at the shoulders, extended at the elbows, the forearms semi-pronated, and the wrists slightly flexed. The posture of the upper extremities is particularly arresting because of its rarity in man. Its occasional presence in the so-called tonic fits of Hughlings Jackson, however, is a fact to which Kinnier Wilson ([18] p. 220) has recently drawn attention. In the same paper this observer has described a state of maintained muscular contraction, following "acute physiological decerebration," in cases of internal hydrocephalus and of gross lesions of the mid-brain, which, as regards its distribution and the resulting

attitudes of the body and its component parts, resembles decerebrate rigidity. The postures of the limbs in some of his cases are reproduced with precision in our quadriplegic patients.

These considerations show that the distribution of the steady muscular tension or rigidity, to which in the absence of movement the maintained posture of the trunk and limbs in Nos. 1 and 2 was due, is at least very similar to those observed in animals after anatomical, and in man after physiological, decerebration. Broadly speaking, the attitude is one of general extension with pronation of the upper extremities or fore-limbs.

Sherrington states that the tone of those muscles which oppose the rigid muscles in decerebrate rigidity is mildly inhibited under the influence of reciprocal innervation. He says: " The tonic excitation is supplied only to one member, the extensor, of antagonistic muscle pairs. There is evidence that the flexors are under a mild tonic inhibition, the counterpart of the tonic excitation of the extensor motor neurons" ([10] p. 105). In other words the tonic or postural reflex, while bringing about steady contraction of the extensors, at the same time causes slight relaxation of the flexors, and the one process is proportionate in degree to the other although of opposite sign.

We made a point of repeatedly examining the condition of those antagonists which from their superficial position are open to palpation, and although our methods did not admit of exact measurements being made some trustworthy information was obtained. In the first place it was certain that the antagonists were less strongly contracted than the agonists. They were more pliable, yielded more to pressure and could be displaced from side to side more readily. In addition they offered less resistance to passive lengthening. We were able, moreover, to satisfy ourselves of the presence of a definite relationship between the states of tonus of antagonistic muscles. The strength of the postural contraction of the rigid muscles varied from time to time, and when the limbs were at rest the stronger the contraction in triceps the more relaxed the biceps and vice versâ. This point was well brought out by a comparison of the states of different antagonists. Some were found habitually to be more relaxed than others and it was noticed that those muscles which showed the least rigidity were opposed in their action to muscles which were invariably strongly contracted. Thus the curve that is given to the tip of the shoulder by deltoid in the healthy individual was absent in No. 1. The muscle was not appreciably wasted, but it was flat and more or less flabby in contrast to the

powerfully contracted and prominent pectorals. On the other hand when the upper limb was at rest triceps was rarely so rigid as the muscles of the anterior axillary wall and biceps usually stood out well, preserved its natural form and was of firmer consistence than deltoid.

Yet it could not be said that any of the antagonists of the rigid muscles which were examined were ever hypotonic; indeed at least some of them always appeared to be more strongly contracted than the corresponding muscles in a healthy individual. Notable examples were biceps, the extensors of the wrist, the hamstrings and the dorsiflexors of the foot. In this class should also be placed the muscles of the anterior abdominal wall which, according to Sherrington ([11] p. 212), exhibit the same reactions as the extensor muscles in the decerebrate animal. That the abdominal muscles subserve postural functions is shown by Kelling's experiments ([5] S. 161) on the intra-abdominal pressure in the dog.

This general but unequal raising of tonicity in somatic muscles when at rest we have also found to hold in spastic cerebral hemiplegia. Here the rigidity is distributed in the trunk and lower limb to the extensor muscles as in the extensor form of spastic paralysis from lesions of the spinal cord, but in the upper limb the flexors and not the extensors are overloaded with tone; hence the well-known attitude of the arm in such cases. But again, examination of the flexors of the lower extremity and trunk and of the extensors of the upper limb shows that these muscles maintain a steady contraction of a strength usually above the normal. As we shall see later, however, the rigid muscles, which by their overbearing activity determine the posture of the affected parts, show reactions which are poorly developed in their antagonists.

For a comparison of the spasticity in the cases under discussion with the rigidity of the decerebrate animal the relationship between the tonic states of resting antagonistic muscle-groups is a question of considerable importance. From laboratory experiments on animals Sherrington ([11] p. 196) has concluded that when one muscle of an antagonistic muscle-pair is contracted the antagonistic muscle is reciprocally relaxed. Our observations on the tonicity of the resting muscles are mainly in agreement with this statement. The muscles which are antagonistic to those exhibiting a high degree of tonic activity were relatively relaxed, but, compared with the muscles of the intact individual, their tonicity was increased.

With regard to the development of spasticity in different parts of

the body there is again considerable correspondence between our patients with quadriplegia and decerebrate animals. In the monkey, dog, cat, rabbit and guinea-pig rigidity after decerebration is more intense in the fore-limbs than in the hind-limbs ([12] p. 319). In the hind-limb the knee is the principal joint affected; the ankle is less implicated but is more rigid in extension than is the wrist in flexion. According to Sherrington the digits are never affected at all.

The condition found in Nos. 1 and 2, examples of quadriplegia, corresponds fairly closely with this description. In No. 1 the rigidity was much greater in the upper than in the lower extremities, although in No. 2 the distribution was less unequal. The extensors of the knees showed more tonic contraction than the extensors of the hips or ankles and, as in animals, the toes could be moved passively up and down without any resistance being encountered. Some of the muscles acting on the fingers, however, were hypertonic and others hypotonic, a condition which Sherrington does not describe in the decerebrate monkey. More especially in No. 1 the thumbs were steadily held against the palmar aspect of the index fingers, this position being maintained by powerful tonic contraction of the adductors and opposers of the thumb. Any effort on the part of the examiner to separate the thumb and index finger was met with strong resistance which increased within limits in proportion to the force exerted and to the rapidity of the attempt at passive displacement. Usually an adductor clonus was set up. But in contrast to the adductor rigidity of the thumb the fingers were extremely flexible. This was especially the case in No. 2 whose fingers could be passively hyper-extended and separated to an extreme degree. Passive flexion at the metacarpo-phalangeal joints could be less completely accomplished and there seemed to be quite definite increase of tone in the extensors of these joints. It will be remembered in passing that of the muscles which play directly on the wrist rigidity was more evident in the flexors than in the extensors.

The little and ring fingers in No. 1 also showed an absence of rigidity and an unusual freedom of movement. But while the other two fingers of the hand, in particular the index, could be readily over-extended, yet sudden jerky movements immediately excited brief contraction of the flexors. In the resting state and under the stimulus produced by slow stretching of their fibres the muscles of the radial fingers seemed to be almost flaccid, although a sudden increase of tension would excite a momentary resistance to passive lengthening.

A feature of postural activity in general, and one which is well

brought out in decerebrate rigidity, is the slow development of fatigue associated with it. If both hemispheres are removed and rigidity is well developed, strong tonic contraction of the extensor muscles is maintained for hours on end. This inexhaustible contractility was present in No. 1, especially in certain muscle-groups, notably the adductors and internal rotators of the shoulders, the pronators of the forearm and the flexors of the wrist. In No. 2, however, spasticity in the upper limbs was never so strong as in No. 1, and was maintained also at a less uniform level. When his arms were supported on the bed triceps usually became almost flabby, but tonic contraction could readily be excited again by passively moving the forearms backwards and forwards so as to alter the tension of the muscle. It should be remembered of course that in our cases conduction between the spinal cord and the cerebral hemispheres was by no means abolished; voluntary power and sensibility though disturbed were still present. This is of interest because in animals with hemi-decerebration, that is to say, with unilateral transection of the mid-brain, a similar waxing and waning of extensor rigidity is usually observed.

Perhaps the most characteristic phenomena of muscles showing postural contraction are what Sherrington has called the "lengthening" and "shortening" reactions. They can be demonstrated in the extensor muscles of the decerebrate preparation in the following way. With the animal lying on his side the observer elevates the hind-limb into the vertical position and the knee remains extended from tonic contraction of vasto-crureus. If he now passively bends the knee, so as to lengthen the extensor, the leg will be held in its new posture by the muscle against the action of gravity, the tonic contraction of the muscle being as strong as before. This is the "lengthening" reaction. So also when the attachments of vasto-crureus are approximated by passively extending the knee, the "shortening" reaction is evoked and the leg is retained in the position into which it has been placed by the same degree of steady contraction of the muscle.

Similar results were obtained from experiments of the same kind carried out on vasto-crureus and triceps in Nos. 1 and 2. In other words, they showed that (1) the steady tonic contraction of these muscles was sufficient to oppose the action of gravity on the part of the limb peripheral to the joint upon which each plays, and (2) this tonic or postural function is independent of the muscle length. This property of muscle in postural contraction to become reflexly adjusted to different lengths while counteracting the same load, led Sherrington to apply the term plastic tonus to what he later termed postural activity.

The conditions essential for the appearance of the shortening reaction are clearly shown in the case of the knee-jerk. When reflex postural activity is much diminished, as it is in the extensor of the knee, in spinal animals ([11] p. 211), and in spinal man ([6] p. 356), the knee-jerk may be brisk and ample, but relaxation is as quick or quicker than contraction, so that the leg falls like a dead weight. When, however, postural contraction is highly developed, as after decerebration, although the initial extensor contraction is just as rapid and easily evoked by tapping the patellar tendon as in the spinal preparation, relaxation is very different. In the decerebrate preparation the leg does not drop at once, but remains in the extended position into which the quick contraction of the knee-extensor has placed it, and is held there for several seconds; then it may gradually fall. A shortening reaction has been appended to the jerk contraction ([11] p. 211).

This phenomenon has been observed in the knee-jerk in spastic paraplegia in extension ([6] p. 357), and we found it to be remarkably developed in the patients described in this paper. In No. 2 it was particularly well shown in regard to both the triceps-jerk and the knee-jerk. The following observation was repeatedly made. The lower limb flexed at the knee and rotated outwards at the hip was passively raised off the bed and held in this position by the observer. Serial knee-jerks were then evoked by gently and rhythmically tapping the patellar tendon at intervals of several seconds. With each jerk the knee became a little more extended than before, and remained in this position until the next reaction was evoked. After several knee-jerks had been excited the position of the limb had become altered from one of flexion to one of almost complete extension. Knee-jerks evoked in this way thus lead to a step-like shortening of quadriceps extensor with progressive extension of the leg. The phenomenon was evoked equally well in No. 2 without the employment of percussion of the tendon. In his case sudden passive stretching of the extensor of the knee often excited a rapid clonus occurring at the rate of about 200 per minute. With each rhythmic movement the leg became extended a little more till finally extension at the knee was complete. The mechanism by which the extensor muscle was shortened in this case was the rapidly recurring excitation of the tonic reflex by the weight of the limb in each relaxation phase of the clonus. Progressive shortening reactions of the same order have recently been studied graphically in the vasto-crureus of the decerebrate cat by Viets ([15] p. 269).

The phenomenon, however, seemed to occur only when the jerks

evoked were of small amplitude. When the reflex was evoked by a strong stimulus so that the upward excursion of the leg was extensive the heel, in the relaxation phase, dropped almost immediately, sometimes to be arrested before it reached the bed. A reaction of this kind could be readily studied in No. 1, in whom extensor rigidity in the lower extremities was less well developed than in No. 2. We can best illustrate the main features of this reaction by describing briefly an experiment which was frequently repeated, and gave more or less constant results. With the patient lying on his back in bed his thigh was grasped by the observer, and his lower extremity suddenly elevated. The increased tension on quadriceps extensor caused by the weight of the leg and foot usually excited the tonic reflex, and the leg remained for a time extended in the air, although the extension was not, as a rule, complete. The patellar tendon or the leg was now smartly tapped so as to evoke a quick vigorous contraction in the extensor of the knee. This brought about sudden elevation of the leg, so that extension at knee became complete, followed almost immediately by rapid fall of the leg and foot from relaxation of the extensor muscle. If the thigh was being so held by the observer that there was considerable flexion at the hip relaxation of quadriceps extensor was usually arrested and replaced by tonic contraction before the heel reached the bed, and after one or two minor jerks the leg came to rest still suspended in the air, but at a lower level than before.

An analysis of the reaction shows that before the patellar tendon was tapped the leg was being supported against its own weight by the postural tonic contraction of quadriceps extensor. The appropriate stimulus for the knee-jerk, differing in its shorter duration and its more sudden application, excited a movement, namely, the upward jerk of the leg, and at the same time inhibited the tonic contraction of the extensor muscle. The quick contraction which caused the jerk was rapidly replaced by relaxation allowing the muscle to be passively stretched by the weight of the leg and foot; and it was only after the muscle had become elongated to more than its original length that the passive stretching excited once more the tonic or postural reflex. The descent of the limb was then arrested and the stoppage again evoked a mild jerk which was repeated before the reaction finally ceased.

The ease with which a tonic or postural reflex is inhibited by a kinetic or movement reflex employing antagonistic muscle groups has been shown by Sherrington and others to be one of the features of postural contraction. The phenomenon can be readily demonstrated in

the decerebrate preparation and in spastic paraplegia in extension in man by evoking a flexion reflex of the lower limb. With contraction of the limb-flexors there is simultaneous and proportionate relaxation of the previously rigid extensors. But in this respect the knee-jerk experiment we have just described is of particular interest, for it shows that in the same muscle inhibition of plastic tonus can occur simultaneously with the excitation of a quick contraction. Moreover the nature of the stimulation required for the postural and the kinetic or movement reactions is of the same kind although differing in intensity and duration. That dissociation of these reflex-effects can occur is most likely due to difference in reaction-times. Attention has been drawn to this difference in relation to the reflex reactions of the movement and postural adductors of the mollusc Pecten, and in this connection Sherrington ([11] p. 204) says that "the speed with which the postural muscle can by contraction shorten its length is relatively slow as compared with that of the movement muscle."

It is worthy of mention again that in our cases it seems that the strength of excitation of the kinetic reflex had some influence in allowing the dissociation to appear, for it was not observed when knee-jerks of small amplitude were evoked by gentle taps on the patellar tendon; then the shortening reaction was immediately appended on the twitch. The probable explanation of the difference obtained with variation in the strength of the stimulus is that, as in reciprocal innervation of antagonistic muscle-groups, the degree of inhibition of postural contraction is proportionate to the degree of excitation of kinetic contraction. Thus with a feeble knee-jerk inhibition of postural tonus in the vasto-crureus is slight, whereas it is more complete with a large knee-jerk.

While dealing with the distribution of rigidity in the resting muscles in the cases of quadriplegia we stated that although the antagonists of the rigid muscles were relatively inhibited yet, as compared with the resting muscles of the intact individual, they were hypertonic. It was also mentioned that certain properties which were conspicuous features of the agonists were poorly developed in the antagonists. These properties are the lengthening and shortening reactions.

When we say that the antagonists were hypertonic we mean that to the clinical tests we employed they behaved in the same way as the more tonic muscle-groups though in lesser degree. This was the case at all events with biceps and the hamstrings. For example, sudden passive stretching of these muscles led to a temporary increase in their tonic contraction, the increment disappearing when the stretching

process was stopped. Thus tonic reactions could to some extent be obtained in biceps and the hamstrings, but they were insufficiently developed to counteract the action of gravity on the limb and the greater tonic activity of the extensors. For in No. 1 when the upper limb was flexed at the elbow so that gravity acted on the forearm the tonic contraction of biceps was never sufficient to retain the forearm in this position and the elbow immediately became extended. The same result was obtained with the flexors of the knee.

As has already been mentioned, however, in No. 2 it was sometimes found that after his upper limb had lain in a flexed position across his chest for some time biceps became the more contracted of the antagonistic muscle-pair. When tested under these conditions the phenomena of well developed plastic tonus could be shown to be present; the forearm could now be placed in any position and held there by the tonic contraction of biceps. Progressive shortening of the muscle could also be induced by evoking a series of biceps jerks, although under the more usual influence of tricipital rigidity this reaction was not obtained.

The occurrence of plastic flexor tone in animal preparations is not unknown. According to Graham Brown ([3] p. 180), states of maintained flexion in the limbs may occasionally appear in the narcotized monkey during systematic examinations of cortical motor reactions. They may also appear from time to time in the decerebrate monkey or anthropoid ape, and maintained flexion of one fore-limb is often found in association with maintained extension of the other. (In our patient flexion of the upper limb occurred sometimes with flexion and sometimes with extension of the opposite upper limb.) Graham Brown notes that the posture of the limb may change either suddenly or gradually, a feature which we observed in No. 2. In No. 2 also, as in the monkey, triceps was in a state of mild relaxation so long as the plastic tonic contraction of biceps continued. In addition Graham Brown has found that in the monkey, which has been decerebrated by section of the neuraxis just in front of the anterior colliculi, unipolar stimulation on the cross section within an area corresponding with the site of the red nucleus evoked flexion of one fore-limb at the elbow with simultaneous extension of the other. These reactions are characterized by a marked after-discharge, so that for several minutes after cessation of the stimulus flexion is maintained on one side and extension on the other.

In hemiplegia with rigidity plastic tonus is usually distributed to

the flexors of the upper extremity, hence the well-known flexed, pronated attitude of the limb. Extensor rigidity is the rule in the lower extremity, but sometimes cases are observed, as in a patient recently under investigation in which the tonic contraction of the flexor predominated and the resting posture of the limb was one of flexion.

Although in No. 2 the upper limbs were sometimes found to be flexed at the elbow from alteration in the distribution of the plastic tonus, the rigidity in the lower extremities was invariably extensor. The flexors compared to the extensors were mildly relaxed ; even when the limb was placed passively in a position of flexion the degree of tonic contraction in the flexors did not perceptibly increase. With the knee in various degrees of flexion experiments were carried out to determine whether the lengthening and shortening reactions could be obtained in the hamstrings. The tests employed showed that they were imperfectly developed and with some of the tests negative results were obtained. For example, when the hamstring tendons were tapped with the lower limb flexed and lying on its outer aspect on the bed a brisk contraction of the muscle was evoked, but the relaxation phase was rapid and even with strong stimulation the leg was not displaced. The elicitation of rhythmic hamstring jerks by repeated taps on the tendons at brief intervals yielded another interesting result : each twitch of the hamstrings appeared to be associated with simultaneous and temporary diminution in the tonic contraction of their opponent muscles, the extensors of the knees. This was the only additional phenomenon to be observed with the first three or four contractions. Corresponding in time to the relaxation phase of each subsequent hamstrings jerk, however, and following immediately on the stage of mild inhibition of the extensors, these muscles were felt to contract suddenly and a quick, short extension movement at the knee occurred. Repeated stimulation caused the leg to extend more and more in short steps.

This paradoxical phenomenon in which the knee is seen to be extended as the result of repeated taps on the hamstring tendon demonstrates the absence of an effective shortening reaction in the hamstring muscle. But this alone would not explain the steplike extension of the knee. It really depends on the operation of post-inhibitory exaltation, the knowledge of which we owe to Sherrington. The depression of one reflex which accompanies the excitation of its antagonist is followed by a phase of increased excitability. Applying this law to our experiment we find that after each of the first three or four taps on the hamstring tendon and the accompanying relaxation of

the vasto-crureus the only phenomenon to be observed is the re-establishment of tone in that muscle. During this time, however, the excitability of the extensor reflex arc had become exalted so that ultimately the re-establishment of extensor tone was in itself a sufficient stimulus to evoke an active shortening reaction of the muscle which was repeated after each subsequent hamstring jerk. Under conditions in which tonic contraction is approximately equally developed in the muscles innervated by antagonistic reflexes this process tends to provoke alternating reactions of opposite phase. For example, so long as stimulation is appropriate, reflex extension will be the inevitable consequence of reflex flexion and vice versa, as in the "mark-time" reflex of the spinal dog and spinal man. Successive induction ([8] p. 213) promotes biphasic activity. But where tonus is developed in one group of muscles in excess over the corresponding antagonists, as in the states of disordered function under consideration, rhythmic sequence of action in opposing muscles is damped down or prevented by the predominant rigidity which overcomes the effect of post-inhibitory exaltation on the reflex arcs controlling the less tonic muscle-groups.

On analysis the resemblance between the rigidity in quadriplegia in extension and the maintained tonic contraction in the decerebrate animal preparation suggests that the two states are closely related. Attention has already been drawn by Walshe ([17] p. 6) to the similarities between the spasticity of hemiplegia and of decerebrate rigidity, and our observations in general support his conclusions.

With Sherrington we believe that tonus is a postural reaction and that, in the states under consideration, it is reflex in origin, the stimulus being provided by tension acting on receptors within the muscles themselves and their tendons. The natural stimulus which evokes the reaction in the trunk and limbs, and so determines the resting attitude of the body, is provided by the tension exerted by gravity, and Sherrington has interpreted the decerebrate rigidity found in extensor or anti-gravity muscles in animals as reflex standing.

Is it reasonable to offer such an explanation for the rigidity or postural contraction in the upper limbs? Man is bipedal and the freedom of the upper limbs from the bondage of locomotion allows them to be used as organs for exploration and prehension. The grasping and conveyance of food to the mouth is probably the elementary function of the human hand and arm. A moment's reflection, however, will show that the upper extremities, although they have ceased to take any direct part in locomotory acts, are still bound to the lower extremities in the

automatic movements of walking and running. In these movements the arms may be brought into action in two different ways; they may perform rhythmic biphasic movements or they may assume postures. The former mode of activity is observed in walking and the latter in running. Thus, when a man is walking quickly along the street he involuntarily swings his arms, the timing, vigour and amplitude of the movements corresponding closely to those of the lower limbs. The arms simultaneously move in opposite directions and the direction of movement of the four limbs at any moment is such that there is a diagonal correspondence. Thus the right upper and left lower limbs are coupled together and swing forwards or backwards at the same time, flexion of one arm occurring with extension of the leg on the same side. The movements of the limbs in walking are therefore the same in man as in four-footed mammals, those of the upper limbs being indirectly locomotory in the former and directly in the latter.

In the act of running, however, postures rather than movements tend to be adopted in the upper limbs. The attitude most commonly observed in a runner going at full pace is one of adduction at the shoulders, flexion at the elbows, flexion of the fingers and extension of the wrists. All the muscles of the upper limbs are in contraction and although to-and-fro movements at the shoulders in diagonal correspondence with the movements of the lower limbs are observed they are usually small.

Let us consider these observations on locomotion in the normal individual in relation with the phenomena we have been investigating. The flexed attitude of the upper limb in relation with the extended lower limb in hemiplegia might be held to have a locomotory significance. This may in part be true, and certainly many hemiplegic patients when walking automatically swing their paralysed arms as they move forward their unaffected lower limbs. But taking into consideration the fact that the hemiplegic patient as a rule recovers the power of voluntary flexion better than that of extension of his upper limb, the rigidity of the flexors is probably more intimately connected with the prehensile function of the limb.

It is otherwise, however, with the upper limbs in quadriplegia in extension. They are kept extended at the elbows and if voluntary power is not abolished extension is stronger than flexion. Let us briefly review again the general attitude of the patient in standing. His lower limbs are straight with his feet apart; he stoops a little forward from slight flexion of his spine and hips; his upper limbs hang down a little

in front of his trunk, extended at the elbows, the forearms somewhat pronated, the wrists slightly flexed; his fingers are extended at the metacarpo-phalangeal joints and otherwise flexed. The whole attitude in its general features is not unlike the position often adopted by the higher apes in standing and suggests in grotesque fashion a static posture in a stage of human development when the upper limbs may have subserved more direct locomotory functions.

Disease or injury of the nervous system results in disturbance of function. There is destruction of activities which in the history of the individual are recently acquired, while those which are older, more organized and automatic are preserved in an altered form. These positive manifestations of disintegrated nervous activity are unusual and abnormal in so far as (1) they are excessive developments of normal phenomena released from the physiological control to which they have been subjected by the intact nervous system; and (2) they present features bearing less resemblance to normal reactions in proportion to the extent to which the functional activities of the part have been acquired. Thus the flexion reflex seen in paraplegia is little more than a crude exaggeration of a normal reaction, especially in infants, belonging to a limb which is largely automatic in its function. On the other hand, the reflex activities displayed by the upper limb in quadriplegic patients may diverge widely and strangely from those usually associated with a part which has been educated up to an infinite variety of specialized movements.

The reactions laid bare by injury or disease cannot be looked upon as exact reproductions of modes of behaviour evoked under similar conditions at an earlier stage of human development. In spite of their modifications, however, they still bear traces of their ancestry, and also of their relationship with analogous reactions in the intact individual.

(B) Significance of "Associated Movements."

Much has been written on the subject of associated movements, particularly by French clinicians; but, as Walshe ([17] p. 16) has pointed out, little attempt has been made to investigate them in the light of modern physiological knowledge. We have studied these reactions in a number of hemiplegic and quadriplegic patients, and believe that our observations justify certain conclusions which seem to throw some light into the obscurity surrounding this subject.

Since reflex reactions of widely different kinds have been erroneously

included under the heading "associated movements," we will define our use of the term in order that there may be as little misunderstanding as possible. "Associated movements" are automatic activities which fix or alter the posture of a part or parts when some other portion of the body is brought into action by either voluntary effort or artificial reflex stimulation. Closely related with such reactions are the muscular activities which occur in the limbs and trunk in association with such automatic acts as yawning, "stretching," and coughing.

The term was originally employed for movements of the paralysed limbs in spastic hemiplegia, which occur with strong voluntary movements of the unaffected limbs. But our observations on reactions of this kind show that the response is not, as a rule, confined to the paralysed half of the body, but also involves the non-paralysed parts. Thus when No. 5, an example of right hemiplegia, attempted voluntarily to bend his left elbow against resistance the following movements took place in his trunk and other three limbs. His right (paralysed) upper limb became adducted and rotated inwards at the shoulder, incompletely extended at the elbow and fully at the wrist and the hand clenched. His trunk became slightly extended from contraction of the muscles on both sides, and his lower limbs stretched out.

A general reaction could be obtained not only when he tried to move one of his non-paralysed limbs against resistance, but also when he made strong voluntary efforts with his paretic upper or lower limb on the right side. For example, with voluntary flexion of his right elbow, the general movement evoked consisted of slight extension of his trunk, flexion of his left elbow and bilateral extension of his lower limbs. Again, voluntary extension of his right (paralysed) lower limb evoked extension of his other three limbs.

But movement is not the main part of associated reactions of this kind, in spite of the prominent place given to it by the majority of previous observers. The most striking feature of an automatic and associated response evoked by strong voluntary effort to displace a part of the body, whether paretic or not, is increased contraction of antagonistic pairs of muscle-groups in the trunk and other three limbs, with the result that strong fixation rather than movement of parts is obtained. Sometimes, but by no means invariably, one group of muscles acting on a joint are contracted more strongly than the opponents, so that movement at this joint results. But such movement is slow, and its appearance seems to be determined by the strength of the voluntary effort made by the patient.

Undoubtedly the earliest and most constant response is increased contraction of all the muscles acting on a joint. This appears first in the muscles of the trunk and of the proximal joints of the limbs, and progressively spreads towards the fingers and toes with increase in the intensity of the exciting stimulus. The associated reaction is always strongest and involves most muscles in the paralysed limbs, and in them appears earlier than in the healthy upper and lower limbs. Thus when No. 5 tried to bend his left (unaffected) lower limb against resistance the associated reaction was observed first in his right lower limb and trunk, then in his right upper limb, and lastly in his left upper extremity. Also, in his trunk and paralysed limbs all the muscles were involved, and movements at different joints were observed at a stage in the reaction when in his left upper limb the muscles of the shoulder only showed increased contraction without actual displacement at this joint.

Associated reactions of the same order as those we have just described in hemiplegia were evoked in the trunk and three limbs in patients with quadriplegia when strong voluntary movements were attempted in the fourth limb against resistance. In No. 2 voluntary power was more impaired in the limbs of the right than those of the left side. Resisted voluntary flexion of the left elbow excited the following response: In the right upper limb there was elevation adduction and backward movement at the shoulder with slight flexion at the elbow and wrist; in the trunk the homolateral rectus abdominalis and contralateral erector spinæ were brought into action more than their antagonists, so that the trunk was rotated to the right; the lower extremities were rigidly extended.

The reaction obtained when the patient attempted to extend his left elbow against resistance was as follows: In the right upper limb adduction and downward movement occurred at the shoulder, extension of the elbow, some pronation of the forearm and slight extension of the wrist; the trunk rotated to the left and the lower limbs became extended at all joints.

Two further observations only will be quoted from our records of this patient's reactions. With strong voluntary flexion of his left knee his right first flexed and then strongly extended with downward movement of the foot; his trunk arched forwards with rotation to the left and his upper limbs rotated inwards and abducted slightly at the shoulders, and flexed at the elbows and wrists.

Attempted voluntary extension of his left knee against resistance

evoked a response in the trunk and opposite lower limb which was very similar to that just described, but in the upper limbs there was adduction with internal rotation at the shoulders, strong extension at the elbows, pronation of the forearms and slight flexion of the wrists.

But, as in hemiplegia, these movements, which under similar conditions varied to some extent even in the same patient in different experiments, did not form the earliest part of the response nor did they necessarily appear, especially in mild reactions. They really were an index of the intensity of the exciting stimulus, namely the voluntary effort of the patient to move one of his limbs. In a reaction the sequence of events was not difficult to follow. If, for example, the patient squeezed an object in his hand all the muscles playing on the wrist, elbow and shoulder were automatically brought into action, although with the exception of extension of the wrist no actual movement need occur at these joints. As he increased his efforts to squeeze the object in his hand the associated response spread from the limb directly stimulated and became manifest first in increased contraction in all the muscles of the trunk and proximal joints of his limbs. The other upper limb and the lower limb on the stimulated side were involved before the crossed lower limb. Still stronger stimulation resulted in spread of the muscular field involved towards the peripheral parts of the limbs and in movements at some of the joints due to greater contraction of some groups of muscles over their antagonists.

Reactions similar to these described above can also be excited by strong protective reflex movements in an upper or lower limb in both quadriplegia and hemiplegia. Thus in No. 2, when the extension reflex was excited in the left upper extremity, the right upper limb became more adducted at the shoulder and slightly flexed at the elbow ; all the muscles of the trunk, but especially those of the left side, became contracted and strong extension occurred in the lower extremities, in the left more than the right.

Again with powerful reflex flexion of the left lower limb there occurred extension of the right lower limb at all joints, strong contraction of the erectores spinæ and to a less extent of the rectus abdominalis and oblique muscles on both sides and bilateral contraction of the abductors and adductors of the shoulder and flexors and extensors of the elbow.

In No. 5, an example of right hemiplegia, the following associated reactions occurred with strong reflex flexion of the right upper limb :

Contraction of all the muscles acting on the left shoulder and elbow joints, although no movement actually took place; all muscles of the trunk and of both lower limbs were brought into action, more powerfully on the right than the left side. Owing to the predominant contraction of the extensor muscles in the lower extremities these limbs remained in an extended position.

With reflex flexion of the right lower limb there occurred contraction of all the muscles of the left lower limb, which remained firmly extended; in the right upper limb contraction of all muscles on both sides of the trunk, strong contraction of abductors and adductors of the shoulder, of the flexors and extensors of the elbow, with a slow and incomplete movement of extension, of the flexors and extensors of the wrist with extension of this joint and of the flexors of the fingers so that the hand became clenched; in the left upper limb contraction of all the muscles of the shoulder and elbow-joints without any movement of the limb.

The remote reflex phenomena we have been describing, whether in association with powerful voluntary contractions against resistance or with strong protective reflex reactions in a limb, possess certain points in common. In the first place, the outstanding feature of the widespread response was increased tone in prime movers and antagonists in regions of the body remote from the stimulated limb. That is to say, although some of the limb segments and the trunk were often displaced the movement was due not to unopposed contraction of certain muscles but rather to an excess of contraction in some muscle-groups over that of their corresponding opponents. Whether a movement occurred or not accession of tone in all the muscles acting on a joint formed the earliest and most constant part of the associated reaction.

Secondly, it was necessary that the protective reflex response or the voluntary muscular effort should be vigorous before actual movements were obtained in the other limbs or trunk, and any displacement was usually of small extent and slow in its development.

Thirdly, these associated reactions invariably appeared earlier, spread farther and were more intense in paralysed parts, although as the result of a strong stimulus they involved healthy regions of the body as well.

Lastly, the muscles first innervated in a limb were those which played on the proximal joints, while any displacement was generally more noticeable in the peripheral segments.

To sum up, our observations seem to show that the phenomena

usually spoken of as associated movements are in reality widespread postural reactions. This conclusion has also been arrived at by Walshe ([17] p. 19), but the prominent feature of these reactions, namely tonic innervation of opposing muscle-groups, has hitherto escaped attention. When one functional muscle-group acting on a joint is innervated more strongly than its antagonists movement of the limb results but it is leisurely and rarely extensive. Movement, however, does not occur and a fixed posture is maintained when the antagonistic muscles are almost equally contracted and balance one another in their action. The muscles mainly involved are those of the trunk and the proximal segments of the limbs. These reactions resemble in their main characteristics those observed in the normal individual when strong voluntary efforts are made to move a limb against a heavy load. Under such circumstances the body is automatically adjusted for static purposes in such a way that the voluntary effort is directed towards one part of the body under the most favourable mechanical conditions, whilst at the same time equilibrium is maintained.

Chapter VI.—Distribution of Innervation and Inhibition in Reflex Movements and Postural Reactions.

Beevor's investigations ([2] p. 54) on muscular action have demonstrated that in regard to their behaviour in a voluntary movement of a limb muscles can be classified in two groups: (1) those which are contracted and (2) those which are relaxed. Of the muscles which show increased contraction some perform the function of prime movers and are directly concerned in bringing about the required movement at a joint while the others carry out subsidiary functions as fixators and synergists. Muscular relaxation is observed in those muscles only which by their action would prevent the desired movement from taking place; in other words they are the antagonists of the prime movers. Thus, when voluntary flexion of the elbow is carried out against resistance, all the muscles acting on the shoulder and wrist joints may be thrown into contraction as fixators or synergists while the inner and outer heads of triceps, the true antagonists of biceps and supinator longus, are relaxed.

When the movement of flexion is confined to the elbow-joint the state of the long head of triceps is of considerable interest. Its behaviour is opposed to that of the humeral parts of the muscle and it contracts as a fixator of the shoulder. Thus, triceps, which is

generally considered as an anatomical unit with one function, namely extension of the elbow, is composed of two parts which may subserve different functions in a flexor movement of the limb. Both parts have a common insertion into the olecranon process and may act together as extensors of the elbow. But the humeral portion is the only pure extensor, and as the true antagonist of supinator longus and the short head of biceps is relaxed in flexion of this joint. The long or scapular portion of triceps crosses the shoulder as well as the elbow-joints, and in voluntary flexion of the elbow may be reflexly brought into action, not as an extensor of this joint, but with long head of biceps to fix the shoulder and so facilitate the short head of biceps in flexing the elbow.

Several other limb muscles, such as biceps and rectus femoris, belong to the same group as triceps, in that each is physiologically speaking made up of two muscles with a common insertion but different origins and under certain circumstances subserving different functions. One part acts on one joint only, the other acting on two, and while both may be contracted or relaxed together under other conditions there may be simultaneous innervation of one and inhibition of the other. Ignorance of this fact has led certain observers to deny the occurrence of muscular relaxation in reflex movements ([14] p. 154).

In voluntary movements of the paretic limbs in quadriplegia and hemiplegia the innervation of muscles directly or indirectly concerned obeys the same laws as those obtaining in voluntary movements in healthy individuals. With increased contraction of prime movers, synergists and fixators there is relaxation of pure antagonists. For example, with voluntary abduction of the arm at the shoulder against resistance the pectorals, which in the resting posture are strongly contracted, undergo relaxation.

The motor taxis in reflex movements of the limbs in decerebrate and spinal mammalian preparations has been analysed by Sherrington [8], who has brought to light most of the known physiological laws of motor innervation. In a movement such as reflex flexion of the fore or hind limb certain muscles become contracted as prime movers, synergists, and fixators, while others, the true antagonists, such as vasto-crureus, are relaxed. The muscles of the first group show concurrent innervation, and those of the second group reciprocal inhibition. Further, the degree of contraction in the fixators and synergists, and of relaxation in the antagonists, is determined by

and proportionate to the amount of innervation of the prime movers.

Our investigations on the examples of spastic quadriplegia and hemiplegia quoted in this paper seem to show that reciprocal innervation of antagonistic muscles is present in some of the reflex movements examined. For example, when the flexion reflex of the lower limb is excited by appropriate stimulation, vastus internus, which with the limb at rest is in strong tonic contraction, becomes relaxed. The tightening of the patellar tendon that invariably occurs is due to the action of the long head of rectus femoris which is employed in the reflex as a flexor of the hip. Sometimes the muscles of the calf contract, although less strongly, than the dorsiflexors of the ankle; but it should be remembered that the only pure extensor of the ankle, namely soleus, is covered over by gastrocnemius, which, by its attachment to the femur, may act under certain circumstances as a flexor at this joint.

In the flexion reflex of the upper limb in hemiplegia the tone of at least some of the antagonists is diminished. The movement at the proximal joints consists of abduction with external rotation of the shoulder and flexion of the elbow. The pectorals thus act as opponents, and since they can readily be palpated, and are in the resting posture of the limb strongly contracted, it is easy to determine that relaxation occurs. After carrying out many investigations in different patients on the state of triceps during a flexor reaction of the upper limb, we arrived at the conclusion that it sometimes became relaxed and at other times its tone seemed to increase. So far as it could be ascertained, the behaviour of the muscle depended upon whether or not movement occurred at the elbow and shoulder. If biceps and supinator longus were brought strongly into action without consequent displacement of the elbow, and if the shoulder remained fixed, then triceps, in part at least, was also innervated. But with strong flexion of the elbow the humeral portion of triceps seemed to relax. It was difficult to be certain what occurred in the extensors of the wrist and fingers when flexion was evoked by scratching the palm of the hand.

Reciprocal relaxation of antagonists to the prime movers could also be demonstrated in the reflex response with flexion of the elbow in Nos. 2 and 4, examples of quadriplegia.

So far, then, it may be said that in those reflex movements which flex or fold up the upper or lower limb in quadriplegia or hemiplegia, those movements which can be evoked from the periphery of the

corresponding limb, there is concurrent innervation of synergists, fixators and prime movers and reciprocal inhibition of at least some antagonists. The motor-taxis evident in these reactions, therefore, corresponds to that in voluntary movements against resistance and in reflex flexion of the fore and hind limbs in decerebrate and spinal animals.

When we come to analyse the distribution of innervation which occurs during the extension reflex of the upper limb in quadriplegia, we are at once faced with the difficulty of ascertaining what takes place in all the muscles antagonistic to the prime movers.

It will be recollected that in the cases of quadriplegia (Nos. 1, 2, and 3) the upper limb presented general spasticity, which was more marked in those muscles determining the resting posture, consisting of adduction and internal rotation at the shoulder, extension at the elbow, pronation of the forearm, slight flexion of the wrist and of the fingers at the interphalangeal joints. This resting attitude was accentuated during the extension reflex with the addition of strong retraction at the shoulder and hyperextension of the fingers. While it was impossible to be certain whether muscles antagonistic to the prime movers were actively contracted or not during the development of the movement, it was comparatively easy to ascertain their condition during the maintenance of the posture produced by the reflex. For example, there was no doubt that deltoid, spinati, and latissimus dorsi were strongly contracted as well as pectorals, and that the tone of biceps increased as well as that of triceps. More difficulty was experienced in determining whether the extensors of the wrist were in a state of tonic contraction owing to the obvious activity of the extensors of the fingers.

There are similar obstacles to precise observation with ordinary clinical methods on the state of certain muscles of the lower limb when its extension reflex is excited, but it is justifiable to draw attention to the resemblance presented by the latter to the extension reflex of the upper limb. In both cases stimulation of the reflex results in accentuation of the already extended attitude of the limb. Although it is not justifiable to state that no muscles undergo relaxation it is certain that the majority of the muscles accessible to palpation, whether fixators or antagonists, are concurrently innervated. The effect of the reflex is in fact more in the nature of the assumption of a posture than of a movement comparable to the withdrawal of the limb brought about by the flexor reflexes.

It should also be noticed that the focus of the receptive field for the extension reflex in both the upper and lower extremities is found at the base of the limb.

Concurrent innervation of antagonistic muscles is perhaps most easily demonstrated in the remote postural reactions which are frequently associated with either voluntary movement against resistance or a strong reflex response in a limb. These reactions have been described in detail in the preceding chapter, and stress has been laid on certain characteristic features observed in them. One of the most noticeable and constant was the simultaneous increase of tone in all the muscles acting on any joint involved in the reaction, irrespective of the tonic state of the muscles at rest.

Sometimes the innervation was apparently almost balanced and the joint was more or less fixed ; at other times, and more commonly in some joints than in others, and depending also upon the strength of the response in the limb directly stimulated, innervation was unequal and displacement occurred from stronger contraction of some muscles than of others. Usually the displacement was slow and did not alter in direction with increase of intensity of the exciting stimulus. But occasionally reversal of movement was noticed. Thus in No. 2, an example of quadriplegia, with increasing voluntary effort to bend the left knee against resistance, slight flexion of the right lower limb usually appeared early but was later replaced by extension at all joints.

An interesting example of a similar condition is provided by the coarse tremor of the hand that was frequently observed in associated postural reactions in the upper limb in quadriplegia and also in the paralysed upper limb in hemiplegia. The posture of the wrist with the upper limb at rest in both cases was slight flexion due to preponderance of tone in the flexor muscles. But in an associated reaction evoked by either voluntary or reflex movement of another limb the distribution of tonus was changed. Contraction in both muscle groups increased, but the accession of tone was greater in the extensors than in the flexors, so that the posture of the wrist gradually changed from one of flexion toward one of extension. A point in the reaction was reached when the slow extension of the wrist was replaced by a coarse tremor, flexion alternating with extension. In the case of the paralysed upper limb in hemiplegia if the associated postural reaction included extension at the elbow and supination of the forearm tremor of these parts was also liable to occur at a certain stage in the response. It could, however, be made to disappear by either diminishing or increasing the intensity of

the reaction, which had the effect of allowing the contraction of the flexors of the wrist and elbow and the pronators of the forearm to be greater or less than their corresponding antagonists. As will become evident, this is a point of some importance in the interpretation of the physiological significance of the tremor.

The probable explanation of this biphasic reaction is found by referring to Sherrington's investigations on rhythmic reflexes of the lower limbs in decerebrate preparations with extensor rigidity ([13] p. 96). His experiment was as follows: Faradic stimulation of either peroneal nerve evoked flexion of the limb and extension of the other lower limb, the antagonists in both cases being reciprocally relaxed. When, however, both nerves were simultaneously excited with faradic stimuli of approximately the same strength relaxation of antagonists disappeared and opposing muscle groups of both knees became contracted. Along with this change from relaxation to contraction in the antagonists the degree of contraction of either the flexor on the stimulated side or the extensor on the opposite side was found to be less than when one peroneal nerve only was stimulated. He was able to show that the innervation of each muscle group under bilateral stimulation was equivalent to the algebraical summation of the two opposing influences. Thus for example the flexor of the right knee was strongly contracted when stimulated through the right peroneal nerve only, but the degree of this contraction was diminished when the left nerve was simultaneously excited, the effect of which as regards the muscle was relaxation. To use Sherrington's term, opposing muscles were under the influence of "double reciprocal innervation." ([13] p. 94]).

By varying the relative strength of bilateral stimuli Sherrington obtained differing results. When stimulation on both sides was so arranged that the innervation of opposing muscles was equal rhythmic alternating movements in opposite phase usually developed. Variations in the strength of the opposing stimuli led to the maintenance of different attitudes of the limbs with contraction of all the muscles.

Considered in the light of these experimental results, it is likely that the rhythmic alternating movements of the hand, forearm and elbow which were observed as part of associated postural reactions in the upper limbs in some of our patients, was a manifestation of the action of two equal but antagonistic forces acting alternately. The stimulus which under this condition started and kept going these biphasic movements was the weight of the segment of the limb

itself, for they tended to appear only when the limb was unsupported. In the muscles of those joints at which displacement occurred in one direction only, concurrent innervation was unequal in antagonists and prime movers.

In our opinion the principle of double reciprocal innervation is also the physiological explanation of the tonic state of muscles at rest. In both spastic quadriplegia and hemiplegia all the muscles of the large joints accessible to inspection and palpation show hypertonicity, but tonus is developed in excess in some more than in others, hence the prevailing attitudes of the limbs and trunk at rest. Here again there is concurrent but unequal innervation of opposing muscles at different joints.

To sum up our conclusions, observations made by inspection and palpation of muscles upon the distribution of innervation in spastic hemiplegia and quadriplegia appear to show that in voluntary movements of healthy and paretic limbs and in reflex flexion movements of the upper and lower imbs there is innervation of prime movers, synergists and fixators and reciprocal relaxation of antagonists.

But in associated postural reactions of the trunk and paralysed or healthy limbs, tonic postures of the body at rest and probably reflex extension reactions of the upper and lower limbs evoked by stimulation of the axilla or groin, all muscles involved show increased contraction and are under the influence of concurrent innervation. These reactions result from unequal innervation of opposing muscle-groups; when this inequality disappears and innervation is balanced, rhythmic movements due to alternating contraction and relaxation of opposing muscles may appear.

General Summary.

In many cases of quadriplegia and hemiplegia reflex movements of the upper limbs can be elicited analogous to those which are known to be present in the lower limbs in cases of paraplegia.

In all the cases of hemiplegia recorded in this paper, with one exception, a flexion reflex of the upper limb was obtained (p. 434).

In all the cases of quadriplegia and in one case of hemiplegia an extension reflex of the upper limb was obtained. In two cases of quadriplegia it was possible, in addition, to evoke a flexion reflex of the upper limb (p. 434).

A flexion reflex of the upper limb usually comprises movements of

abduction and external rotation at the shoulder, of flexion at the elbow, wrist and finger joints. Its receptive field includes the palmar aspect of the hand and fingers, the inner side of the forearm and arm, the walls of the axilla and the upper part of the chest. The focus of the receptive field lies in the palm of the hand (p. 434). Appropriate stimuli are of a potentially painful nature, such as scratches or pinches. The response was uniphasic in hemiplegia, and biphasic in one example of quadriplegia (p. 435).

An extension reflex of the upper limb comprises movements of elevation, retraction, adduction and internal rotation at the shoulder, extension at the elbow, pronation of the forearm, flexion at the wrist and hyperextension and adduction of the fingers and thumb (figs. 2 and 3). The receptive field is co-extensive with that of the flexion reflex but its focus lies in the axillary walls (p. 432). The appropriate stimuli are similar to those of the flexion reflex (p. 428).

The spasticity of patients with quadriplegia in extension and hemiplegia presents close analogies with that of the decerebrate anthropoids described by Sherrington (p. 450).

The so-called "associated movements" are postural reactions; they were found in all the cases of quadriplegia and hemiplegia under consideration accompanying strong reflex and voluntary movements (p. 436).

In all the cases of quadriplegia and hemiplegia we have described voluntary movements and flexion reflexes of the upper and lower limbs showed the phenomenon of reciprocal inhibition of antagonists. Concurrent innervation is a feature of all postural reactions of the trunk and limbs, whether at rest or secondary to strong reflex and voluntary movements, and probably of the extension reflexes of the upper and lower limbs (p. 468).

Appendix.

Case 2.—A. A., aged 29. Following an attack of "influenzal meningitis" at the age of 1 year and 9 months the patient is said to have been weak in both lower limbs and to be troubled with involuntary movements of his head.

When 6 years old he was able to walk to school, a distance of half a mile without assistance, but he was unable to run. His progress at school was up to the average standard for his age.

He left school at 15 and went to help his brother, who carried on a grocer's business, and, although walking was difficult for him, he canvassed the houses in the neighbourhood for orders. After a year he gave up this employment to enter Beck's Optical Works in Kentish Town. He was greatly handicapped, however, by weakness and clumsiness of his movements and his tendency

to fall, and at the end of two years his employers discharged him. Since 1912 he has done no work and thinks he has got more helpless.

Since 1919, when some of the tendons of his feet were divided, he has been mostly confined to bed.

He came under our observation in December, 1920.

There is nothing worthy of note in his family history.

Summary of his Condition between December, 1920 *and December*, 1921.

He is moderately intelligent and is reconciled to his disability. He has never suffered from fits or convulsions: speech and articulation are normal. In the right ear he hears the ticking of a watch at a distance of six inches, but he is completely deaf in his left ear. His visual fields are full and his fundi are normal. His left pupil is slightly larger than his right, but they are both central, regular in outline and react well to light and on convergence. Ocular movements are full; there is no diplopia or strabismus, but nystagmoid movements are evoked by lateral fixation to either side. Sensibility to light touch and pin-prick appears to be relatively diminished on the right half of his face, except his nose. In other respects the functions of his cranial nerves are normal.

Motion: voluntary power.—All movements at the different joints can be carried out but they are decidedly weak. The left upper limb is stronger than the right; in both backward movement and adduction at the shoulder, extension at the elbow, pronation of the forearm and flexion at the wrist are stronger than the movements in the opposite directions. The thumb can be approximated to the tip of each finger, but its terminal phalanx is kept hyperextended and the movements are clumsy.

Extension of the trunk is stronger than flexion and he is unable to sit up in bed without assistance.

His right lower limb is weaker than his left; in both the weakest movements are flexion and adduction at the hip, flexion at the knee, and dorsiflexion and eversion of the foot.

The voluntary movements of his neck are not obviously impaired.

Involuntary movements.—There are occasional slight flexor movements of his lower limbs, more often on the right side. Involuntary movements of his upper limbs, apart from the associated reactions about to be described, have not been observed.

Postural reactions associated with voluntary movements.—With voluntary flexion of his left elbow against resistance the following displacements were observed: In the right upper limb adduction and backward movement at the shoulder and slight flexion at the elbow and wrist; in the trunk rotation to the right; in the lower limbs bilateral extension with downward movement of the feet and toes.

With resisted voluntary extension of the left elbow there occurred downward

movement and adduction at the right shoulder, strong extension at the elbow, pronation of the forearm, and slight extension at the wrist ; the trunk rotated slightly to the left and there was bilateral extension of the lower limbs.

When the left lower limb was flexed against resistance, the movements in remote parts were slight flexion followed by extension of the right lower limb, rotation of the trunk to the left, and extension with adduction and inward rotation of both upper limbs.

Voluntary extension of the left lower limb against resistance evoked a general reaction which was approximately the same as that just described, In each of these reactions antagonistic muscles were contracted.

Gait.—With the aid of jointed mechanical supports for his lower limbs fitted to a spinal jacket, he was able to walk if helped to maintain his equilibrium.

Tone.—All the muscles acting on the large joints of the upper and lower limbs (except deltoid on the right side) and those of the trunk from the shoulder girdles downwards were spastic, but the right upper and lower limbs were more affected than those on the left side. Spasticity was developed in some muscle-groups more than in the corresponding antagonists. The more tonic muscles were the retractors, adductors, elevators and internal rotators of the shoulders, the extensors of the elbows, the pronators of the forearms, the flexors of the wrists and the extensors of the trunk and lower limbs, including the calf muscles. The fingers and toes with the exception of the adductors of the thumbs were hypotonic. The general attitude of the body at rest was one of extension with internal rotation of the upper limbs.

Sometimes after an upper limb had been placed in the position of flexion at the elbow biceps became more tonic than triceps.

The tests employed in the investigation of spasticity and the results obtained are described in the text in Chapter V.

Reflexes—The application of potentially painful stimuli to the palmar aspect of the fingers and hand, the inner and flexor aspects of the forearm or upper arm, the axillary walls or the upper part of the chest from the second to about the sixth ribs in front and behind, evoked a movement of the upper limb which was almost identical with that described in No. 1. The inner walls of the axilla were found to be the focus of the receptive field. Alteration in the situation of the stimulus within the receptive field had little effect in modifying the form of the response. By firmly pinching the tissues of the hypothenar eminence or of the forearm a quick movement of abduction at the shoulder and flexion at the elbow followed by abduction and extension could sometimes be obtained. Simultaneous stimulation on both sides elicited a bilateral biphasic response.

The abdominal reflexes were present, but diminished.

Stimulation of the sole of the foot and of the leg below the knee evoked reflex flexion of the lower limb, including upward movement of the toes. The threshold value of stimulus was lowest in the sole of the foot.

Extension at hip and knee and downward movement of the foot and toes followed appropriate stimulation of the thigh. The focus of the receptive field for the extension reflex of the lower limb was the upper part of the inner aspect of the thigh, the external genitalia and the perineum. When the nocuous stimulus applied to the thigh was intense, reflex flexion instead of reflex extension of the lower limbs was usually obtained.

The tendon reflexes of the upper and lower limbs were very brisk and knee and ankle clonus could be evoked.

Postural reaction associated with the extension reflex of the upper limb.—In the opposite upper limb there usually occurred contraction of all the muscles of the shoulder and elbow joints with some adduction at the shoulder and flexion at the elbow.

The flexors and extensors of the trunk were brought into action but more on the side of the stimulated upper limb.

The lower limbs remained rigidly extended at hips and knees, with some downward movement of the feet and toes. There was increased contraction of all the muscles, but the reaction was stronger in the ipsilateral lower limb.

Postural reaction associated with the flexion reflex of the lower limb.—The associated response appeared first in the opposite lower limb, and as in all reactions of this kind, antagonists, as well as prime movers and fixators, were brought into action. The limb remained extended at all joints. In the trunk there was strong contraction of the extensors, and to a less extent of the recti abdominales. The upper limbs became more adducted and rotated inwards at the shoulders, extended at the elbows and flexed at the wrists, with the fingers hyperextended; all the muscles at these joints, however, showed increased contraction.

Sensation.—The patient did not suffer from pain or other abnormal sensations. Sensibility to light touch was impaired on both sides below the level of the third cervical root area ; the defect was greatest in the area of distribution of the fourth, fifth and sixth cervical roots, but was nowhere complete. Sensibility to pain and to heat and cold was abolished on the left side (except over the buttock) below the third cervical root area, within which painful stimuli evoked an over-reaction. On the right side sensibility to these stimuli was less affected except on the outer aspect of the upper limb and on the upper part of the chest above the second rib. Postural sensibility was grossly affected in both upper limbs, but the loss was less severe in the lower extremities.

Bladder and rectum.—There was impaired voluntary control over the bladder and rectum.

Vasomotor, sudorific and nutritional changes.—The patient was thin but there was no localized wasting, except of deltoid on the right side, which failed to respond to faradic stimuli. Gross vasomotor and sudorific disturbances were not observed.

Subsequent history.—This patient was attacked with acute lobar pneu-

monia which proved fatal. The post-mortem examination on January 5, 1922, showed incomplete, apparently traumatic, healed, transverse myelitis of the fourth and fifth cervical segments of the spinal cord.

Dr. Hubert M. Turnbull furnished the following preliminary report of the autopsy for the purposes of this article :—

There was a band of degeneration in the fourth cervical segment crossing dorsal to the canal, destroying the whole of posterior columns save two isolated dorsal areas, and dipping ventrally for a short distance into each lateral column. In the upper and central thirds of the fifth cervical segment a cross section of cord rendered trefoil by wedges of gelatinous gliosis passing inwards and ventralwards from region of posterior roots; the ventral third of the fifth cervical segment was split into some seven areas by strands of gelatinous gliosis containing minute cavities. Secondary ascending degeneration was seen in the ventral portion of the column of Goll above the fourth cervical segment; descending degeneration in dorso-lateral tracts below the fifth cervical segment. Punctate hæmorrhages were frequent in the centre of the posterior columns of the sixth thoracic segment. Dense adhesions between arachnoid and dura were present over the fourth and fifth cervical segments; considerable adhesion over the remaining cervical segments. Slight adhesion and conspicuous vascularity over the remainder of the inner surface of the dural theca. Conspicuous opaque, white thickening of the leptomeninges over the fifth and seventh thoracic segments. No thickening of leptomeninges of brain and brain-stem. Antero-posterior mobility between the bodies of third and fourth cervical vertebræ was greater than in the remainder of neck, but there was no definite evidence of previous fracture-dislocation. Cavity of the left middle ear and mastoid cell narrowed by chronic osteitis. Perforation of left drum. Mass of wax in left external auditory meatus. Scoliosis with convexity to the right in the mid-thoracic spine. Considerable lordosis. Extreme lateral compression of thorax (chicken-breast). Tapered fingers most conspicuous on the right hand. Œdema of the feet and legs. Wasting of the right thigh; great wasting of the left thigh.

Case 3.—Gunner W. J., R.F.A., aged 29. On July 15, 1916, he was wounded by pieces of shell casing. Two small wounds were found to the left of the middle line behind at the junction of his neck and shoulder and a third on the outer side of his left arm. Consciousness was retained and he believed he had full control over his bladder and rectum, but for a time his upper and lower limbs were completely paralysed. Two hours after the injury his right upper and lower limbs had begun to recover.

He was evacuated to England and on July 18 was admitted to King George's Hospital, London.

Condition on admission: He was dull and heavy, answered questions slowly and inaccurately and obviously resented examination. His temperature and pulse rate were slightly raised and he complained of headache.

The left pupil and palpebral fissure were smaller than those on the right side. There was complete paralysis of the left upper limb with the exception of slight contraction on voluntary effort of biceps and deltoid. In the right upper limb voluntary extension and flexion of the wrist and fingers could be carried out feebly.

Voluntary contraction of the rectus abdominalis was observed on the right but not on the left side.

All voluntary movements were present in the right lower limb and were fairly strong, but the left lower limb was completely paralysed.

There was complete analgesia to pin-pricks on the right side below the level corresponding to the sixth cervical segment and on the outer aspect of the left upper limb within the area supplied by the sixth cervical segment. Sensibility to heat and cold was abolished on the right side within the analgesic zone. Recognition of changes in passive position was defective in the toes of the left foot only and in the right lower limb vibrations of a large tuning-fork (C_{128}) were not appreciated. Sensibility to cotton wool was not impaired.

The tendon reflexes in both the upper and in the left lower limbs and the abdominal reflexes on both sides were abolished. The extensor form of plantar response was evoked on either side.

The left lung was dull to percussion behind and in the axilla, vocal fremitus was absent and the breath sounds were feeble. The patient was troubled with a cough and there was much expectoration.

It was reported after X-ray examination that a small fragment of metal had passed between the fifth and sixth cervical vertebræ in the region of the lateral processes on the left side; the adjacent surfaces of the vertebræ were damaged. A second fragment had travelled between the sixth and seventh cervical vertebræ.

Operation performed by Lieut.-Colonel Armour on September 1, 1916.—The spinous processes of the injured vertebræ were exposed. A piece of metal was found lying on the top of the left lamina of the seventh cervical vertebra. The laminal arch of the fifth cervical vertebra was found to be damaged and a piece of bone had been driven on to the dura. The dura was adherent to the fifth and sixth cervical vertebræ and when the membrane was opened the cord was found to be adherent to it at this level. A small piece of metal was lifted from the cord on the right side opposite the fifth cervical vertebra.

Summary of Condition up to June 26, 1917.

On September 12, 1916, the operation wound was healed, and the area of sensory loss in the left upper limb had diminished and voluntary movement of the arm had increased.

On January 29, 1917, he had full control over his bladder and rectum.

On March 13, 1917, the patient began to have involuntary extensor spasms of both upper limbs, which tended to appear as he dropped off to sleep. Voluntary power in his right upper limb was practically normal and had

improved in his left upper limb at the shoulder and elbow. His left hand when at rest was held in a position of flexion at the wrist, hyperextension at the metacarpo-phalangeal joints and flexion at the interphalangeal joints.

It was found that when the skin in the right pectoral region was scratched a reflex movement was obtained in the left upper limb, consisting of extension of the fingers, flexion of the wrist, and extension of the elbow. The reaction could be obtained by similar stimulation on the left side. When the stimulus was strong it evoked a flexor spasm in the left leg in addition to the movements in the left upper limb.

On scratching the sole of the left foot there occurred reflex flexion of the left lower limb with upward movement of the foot and toes and extension of the right knee with upward movement of the foot and toes. On scratching the sole of the right foot reflex flexion of the right lower limb and extension of the left lower limb with downward movement of the foot and toes were obtained.

Later observations of the extension reflex of the left upper limb showed that the reaction included adduction and internal rotation of the shoulder and adduction of the thumb with flexion of its terminal phalanx. There was also adduction of all the fingers. The receptive field of the reflex response, which was strictly confined to the left upper limb, was bilateral and included the palmar aspect of the fingers and hands, the flexor and inner aspects of the forearms, the inner aspects of the upper arms, the walls of the axillæ and the upper part of the chest in front and behind between the second and fifth ribs.

Recovery of voluntary power continued and in May, 1917, he was able to flex and extend his fingers, thumb and wrist on the left side and to pronate and supinate his left forearm. Voluntary movements at the elbow and shoulder had improved. In the left lower limb all movements at hip and knee were good and he was able slightly to move his toes up and down, but movements at the ankle were absent.

Case 4.—A. B. C., aged 19. Jewel-case maker. Came under observation on February 14, 1921.

Three years ago he suffered from an illness of three weeks duration and diagnosed as "influenza." When he began to get about he noticed weakness and stiffness of both lower limbs on the right more than the left side. The disability slowly increased, and a year later his left upper limb became affected in the same way. At this time he began to suffer from sharp stabbing pains in his lower limbs, chiefly in the anterior aspects of the thighs and behind the knees. The pain was intermittent, and was aggravated by cold and wet weather and by fatigue. There were also occasionally pricking pains in his fingers and arms, which were associated with involuntary abduction movements of his upper limbs, and any object that he happened to be holding in his hands at the time was thrown to the ground.

During the past year his disability has been stationary.

Previous history.—He had measles as a child, and pneumonia at the age

of seven. He was liable to attacks of bronchitis each winter up to the age of fourteen years.

His family history reveals nothing of interest in connection with the case.

Summary of Condition at the Present Time.

He is bright, intelligent, and gives a good account of himself; he sleeps well, and appears to suffer little discomfort. Speech and articulation are normal. He has never suffered from fits or convulsions. Occasionally frontal headaches trouble him, but they are never associated with vomiting. Hearing and vision are good on both sides, and his discs and fundi are normal. Apart from nystagmoid movements on extreme lateral fixation of his eyes to the right or left the functions of his cranial nerves are normal.

Motion: voluntary power.—He is able to perform all voluntary movements with his trunk and limbs, but they are weak; his left upper limb is more affected than his right.

In the upper limbs voluntary movements of extension are weaker than those of flexion, and the opposite is the case in the lower limbs.

Involuntary movements.—Involuntary movements of extension of his lower limbs sometimes occur in the morning when he wakes. As previously mentioned, involuntary quick extension of an upper limb is liable to follow the presence of sharp, pricking pains in the fingers and arm, to which he is sometimes liable. Knee-clonus, unilateral or bilateral, is frequently observed when the patient lies in bed; it occurs particularly when the patient is excited for any reason.

In the morning when he yawns on waking, his upper limbs become extended above his head, and this posture tends to persist for some time after the act of yawning has ceased. As part of this automatic act the trunk and lower limbs are rigidly extended.

Tone.—All the muscles of his limbs, trunk and shoulder-girdle, including trapezius on both sides, are spastic. The degree of spasticity, however, is not excessive, but is unequal in its distribution; thus the lower limbs are more affected than the upper limbs, and the left more than the right upper limb.

The muscles of the shoulder-girdle are all affected equally, but the flexors of the elbows, the pronators of the forearm, the flexors of the wrist, and the extensors of the trunk and lower limbs are more hypertonic than their corresponding opponents.

Nutrition.—The patient is thin, but there is no local wasting in any part of the body.

Gait.—He is able to walk stiffly without support. In walking his right knee is, as a rule, slightly bent, and he walks on the balls of the toes of his right foot. His left lower limb is rotated inwards.

Reflexes.—All the tendon jerks in the upper and lower limbs are much increased, and are equal on the two sides.

There is bilateral knee and ankle clonus. The clonus, when present in one lower limb, can be immediately arrested by evoking either a flexion reflex

in the same or opposite lower limb. In the latter case the flexion reflex is associated with increased tone in all the muscles of the crossed lower limb, but mostly in the extensors.

The abdominal reflexes are present in all segments, but are brisker on the right than on the left side.

Reflex flexion of the lower limb can be evoked by stimulation of the sole of the foot or of any part of the leg below the knee. The outer part of the sole of the foot forms the focus of the receptive field for the reflex. Stimulation of the thigh of mild intensity usually excites extension of the limb on the same side. The focus of the receptive field for the extension reflex lies in the region of the groin, perineum and external genitalia.

Both reflex flexion and reflex extension are accompanied by extension of the crossed lower limb with downward movement of the foot and toes, although tone in all the muscles of the limb is increased.

Reactions evoked by stimulation of the upper limb or upper part of the trunk.—Gentle stimulation of the palm of the hand, the inner aspect of the upper limb, the walls of the axilla or the upper part of the chest, in front or behind, evokes the following reaction, which varies much in detail, according to the locality of the stimulus within the receptive field. The general response consisted of a quick movement of elevation, adduction with external rotation at the shoulder, flexion at the elbow, and extension of the wrist and fingers. There is usually a slight delay between the application of the stimulus and the appearance of the reaction and relaxation, after the primary movement has occurred, is relatively slow. The response is most easily excited by stimulation of the inner aspect of the upper arm and especially of the walls of the axilla. In this region a gentle moving contact with the tip of the finger, and sometimes a mere touch, is sufficient to excite a reaction.

There is great adaptability in the form of the response to the locality of the stimulus. Thus when the reaction is evoked from the palmar aspect of the hand or fingers, the main part of the reaction consists of extension of the fingers and hand, adduction of the thumb and flexion of the elbow. When the stimulus is applied near the elbow, abduction and internal rotation at the shoulder with flexion at the elbow are prominent. Extension at the elbow, however, is invariably obtained from stimulation of the skin behind this joint. When the stimulus is applied to the inner aspect of the upper arm or to the axilla, two main forms of response are obtained. Adduction and external rotation at the shoulder form the main part of the movement with stimulation of the front part of this region, and abduction and internal rotation when the point stimulated lies within the posterior part of this area.

The outer aspect of the upper limb is also responsive but to a less extent than the inner aspect. Scratching the skin over the deltoid, for example, evokes adduction, strong elevation and external rotation of the shoulder.

Reactions of the upper limb on the same side are also evoked by stimulation of the trunk. Pinching the skin over the upper abdominal wall or lower ribs in front evokes a movement in which the trunk is bent towards the stimulated

side. When the point stimulated, however, is in the neighbourhood of the lower border of the fold formed by pectoralis major, this lateral movement of the trunk tends to be accompanied by a response in the upper limb on the same side consisting of adduction and external rotation at the shoulder, slight flexion at the elbow and extension of the wrist. When the stimulus is applied to the supraclavicular region the same reaction in the upper limb is obtained but elevation of the shoulder is prominent. Stimulation of the skin over the scapula evokes retraction of the upper limb and when the stimulus is applied near the inferior angle of the scapula there occurs, in addition, downward movement of the shoulder, flexion at the elbow, and lateral displacement of the head to the stimulated side. When the situation of the stimulus lies over the upper fold of trapezius near the neck the response consists mainly of elevation and forward movement of the shoulder.

By scratching the skin in the middle line over the upper thoracic spinous processes a backward twitch of the head is evoked along with a bilateral quick movement of elevation of the shoulders. By moving the stimulus downwards to the inter-scapular region in the middle line, the elevation of the shoulders and backward movement of the head is now accompanied by quick backward movement of the shoulders followed by forward movement of these parts.

Motor phenomena in association with reflex flexion of the lower limbs.—Associated reactions are not observed with nociceptive reflex movements of the upper limb but with reflex flexion of the lower limb the following general response is usually obtained. The crossed lower limb is strongly extended at hip and knee and the foot and toes move down. All the muscles of the limb, however, are brought into action, as also are the muscles of the trunk ; in the upper extremities movement as a rule does not occur, but all the muscles acting on the shoulder and elbow-joints on both sides show increased tone.

Motor phenomena associated with strong voluntary movements.—When the patient makes strong efforts to extend his left elbow against resistance, slight abduction is observed at the right shoulder, flexion at the elbow, supination of the forearm, and extension of the wrist ; the trunk and lower limbs are extended. All the muscles are brought into action, movement at different joints being determined by stronger contraction of some muscles over their corresponding opponents.

With voluntary flexion of the left elbow against resistance, all the large muscles of the right upper limb become more tonic, but very little movement occurs ; the arm is held a little abducted and internally rotated at the shoulder, the elbow is held in a position of flexion or extension, the posture being determined by the position of the joint before the response is evoked ; the forearm is supinated to a slight extent and the hand is extended ; the trunk is often slightly bent forward, and the lower limbs are strongly extended.

Similar reactions are evoked in association with voluntary flexion or extension of the right elbow against resistance.

Strong voluntary flexion or extension of one knee against resistance also evokes a general response in the rest of the body. The knee of the opposite

lower limb usually becomes slightly flexed, although all the muscles are tonically contracted as are also those of the trunk, shoulder-girdle and elbow-joints.

Sensation.—Sensibility is unimpaired, except for defective recognition of vibrations of a large tuning-fork in the lower limbs and of changes in passive position in the toes.

Bladder and rectum.—There is definite frequency of micturition, sometimes associated with hesitancy. Aperients have to be given daily to keep his bowels open.

Vasomotor and nutritional functions are normal.

X-ray examination of the spine shows no abnormality.

Case 6.—L. S., a man, aged 27, was admitted into St. Thomas's Hospital on August 26, 1921.

During the previous week he had experienced a certain amount of abdominal discomfort. On the day of admission he complained in the afternoon of some stiffness of his neck, but continued at his clerical work. After tea he felt unwell and fainted and a few minutes later began to lose power in his right hand and arm and then in his right leg. Twenty minutes later he was deeply unconscious, vomited two or three times, and was brought into hospital.

On examination in bed he was found in a comatose condition with a full and regular pulse and deep regular respiratory movements.

The left limbs presented some resistance to passive movements, but the right arm and leg were completely flaccid. The reflexes on the right side were of the usual hemiplegic character, those on the left being normal.

Three days later the patient was semi-conscious, but more restless and irritable under examination. Cerebrospinal fluid withdrawn by lumbar puncture was under increased pressure and blood-stained.

The Wassermann reaction in the blood and cerebrospinal fluid was negative. The cerebro-spinal fluid contained many blood-cells and a slight excess of lymphocytes. Cultures of the fluid proved sterile.

During the next week or two the patient gradually recovered consciousness, but was found to suffer from a considerable degree of motor aphasia and also from severe headache, which gradually grew less intense.

The cerebrospinal fluid was examined on several occasions, and showed a progressively decreasing amount of blood as time went on. Voluntary power together with an increasing degree of rigidity developed in his right limbs, and by the middle of October he presented a typical picture of spastic right hemiplegia.

Posture.—The right upper limb was held in a position of adduction and slight internal rotation at the shoulder, flexion at the elbow, semi-pronation and flexion at the wrist and flexion at all finger-joints: the right lower limb was in a position of general extension at all joints.

Tone.—There appeared to be a general increase of tone throughout the muscles of the right upper limb with distinctly more tone in the adductors of the

shoulder, flexors of the elbow, and pronators and flexors of the wrist and fingers. In the right lower limb the amount of tone was not so pronounced but was relatively greater in the extensors than in the flexors.

Reflexes.—All tendon-jerks in the right upper and lower limbs were greatly exaggerated. The right abdominal reflexes were less brisk than those on the left side. Stimulation of the right foot produced a general flexor response of the right limb accompanied by an upward movement of the big toe.

A nocuous stimulus, such as the scratch of a pin or a firm pinch applied to the palm of the right hand or to any part of the inner aspect of the right arm and forearm or to the anterior wall of the axilla, evoked a contraction in the right deltoid, infraspinatus, biceps and flexors of the wrist. The contraction of these muscles produced slight abduction and external rotation at the shoulder, flexion and supination at the elbow and slight flexion at the wrist, and was sometimes associated with extension of the thumb. It was noticeable that the contraction of the above-mentioned muscles was accompanied by relaxation of their antagonists.

This reflex was not strong enough to produce great displacement of the segments of the limb and it was not associated with any definite movements in the trunk or other limbs.

Associated postural reactions.—(1) Reflex flexion of the right lower limb, produced by stimulating the sole of the foot, was associated with slight extension at the left hip and knee, with contraction of the right abdominal wall, and with a movement of the right upper limb, which consisted of an increase of internal rotation at the shoulder, slightly more pronation and flexion at the elbow, and some flexion of the fingers.

(2) When strong voluntary movements against resistance were performed by the right upper limb, such as flexion of the fingers, flexion and extension of the elbow, and adduction and abduction of the shoulder, a number of associated movements took place in other parts of the body. There was general bilateral contraction of the trunk and neck muscles, including both trapezii and all the muscles of the abdominal wall. In the left arm there was observed abduction and internal rotation at the shoulder, flexion at the elbow, pronation of the forearm, with strong flexion of the wrist and fingers. In the lower limbs there was a slight definite movement of flexion on the right side and one of extension, chiefly at the proximal joints, on the left side.

(3) Stimulation of the sole of the left foot produced a downward movement of the left big toe, with slight dorsiflexion of the ankle, and slight flexion of the knee. This was sometimes associated with adduction and retraction of the right shoulder.

(4) Powerful voluntary movements against resistance carried out by the left arm or either leg were always associated with certain movements of the right upper extremity, and these consisted of abduction and internal rotation at the shoulder, flexion at the elbow, slight pronation of the forearm, and flexion of the wrist and fingers. If the effort was sufficiently powerful, contraction took place in the trapezii of both sides as well as in other trunk

muscles, and these movements were associated with slight flexion of the right lower limb, and slight extension of the left.

It was noticeable that in these associated movements the contraction of the prime movers was accompanied by a less powerful contraction of their antagonists.

Case 7.—A. E., a man, aged 28, was admitted to St. Thomas's Hospital, on October 13, 1921, complaining of pain in the left side of his head, general weakness and attacks of transient blurring of vision of a few months duration. There was a history of occasional nocturnal incontinuence of urine and of defective memory and attention.

On examination there was found gross papillœdema in both eyes, slight weakness of the right side of the face and of the right grasp associated with very slight impairment of postural sensibility in the right hand. The reflexes were generally normal and what changes were observed from time to time were variable and indefinite.

On November 2, 1921, Mr. Sargent explored the left fronto-temporal region of the brain and found a deep-seated tumour in the substance of the temporal lobe about one and a half inches behind the Sylvian point. No attempt was made to remove the growth, which had the appearance of an infiltrating glioma.

A post-operation right hemiplegia gradually cleared up, but on November 14 the patient became semi-comatose and had general convulsions chiefly affecting the right arm and leg. These recurred at intervals during the next few days and finally left him with a complete flaccid right hemiplegia.

On November 15 the right upper limb was completely paralysed and toneless. No tendon-jerks could be obtained either at the elbow or the wrist. Pinching or scratching the inner surface of the hand or forearm evoked a movement of abduction and external rotation at the shoulder, the movement being produced by contraction of the deltoid and infraspinatus muscles.

On November 22, a similar stimulus provoked in addition a slight contraction of the biceps with a movement of flexion at the elbow and supination of the forearm.

A week later these reflex movements were obtained with greater ease, and in addition to the movements described above there was observed slight flexion at the wrist. The whole limb was still completely lacking in voluntary power, in tone and in tendon-jerks.

If the patient carried out powerful voluntary movements of the left arm against resistance, they were accompanied by slight flexion of the right elbow and perhaps slight pronation of the right forearm.

At the end of December the right upper limb exhibited a slight amount of muscular tone and the tendon jerks could be elicited. Slight voluntary movements were possible at the shoulder and elbow, and the flexion reflex movements included well-marked flexion at the wrist.

Case 8.—W. S., a man aged 58, who was admitted into St. Thomas's Hospital on March 26, 1921.

He gave a history of fairly good health until the last two years, during which he has suffered from sharp intermittent pains in the lower extremities, and from a certain amount of difficulty in walking steadily. More recently, according to his wife, his memory and general intelligence appeared to be failing.

On March 25, 1921, after a restless night, he was discovered in the morning unable to speak, and with weakness of his left limbs.

On admission to the hospital he looked ill and somewhat emaciated. He lay quietly in bed, and could answer questions slowly, but with defective articulation. There was complete incontinence of both sphincters. The head and eyes were turned somewhat to the right, and the pupils were very small and immobile; both upper lids showed a certain amount of drooping and the patient appeared unable to turn his eyes to the left. The left side of the face was weak when compared to the right. The left upper extremity was also very weak, the muscles of the shoulder and elbow being somewhat spastic, those of the wrist and hand flaccid. The left lower extremity was weaker than the right, especially in regard to all flexor movements. There was some impairment of sensibility to both deep and superficial painful stimuli in both lower extremities.

Reflexes.—The arm-jerks were brisk and equal on the two sides. The abdominal reflexes were absent on the left side. Both knee and ankle jerks were not obtained, and a plantar response on the left side was of the extensor type, that on the right side flexor.

Cardio-vascular and respiratory systems presented no marked abnormality.

The tongue was heavily coated, and the abdomen apparently normal.

On March 27 a lumbar puncture was performed and clear cerebrospinal fluid removed. The examination of this fluid showed that it gave a positive Wassermann reaction, and contained a large excess of lymphocytes. There were some increase in the albumin content.

Although treated with intramuscular injections of mercury and two intravenous injections of N.A.B., the patient showed little sign of improvement, became more incoherent and drowsy, and died on April 13.

While under observation it was noticed that stimulation of the inner aspect of the left arm and of the palm of the left hand by means of pricking or a scratch evoked a reflex movement of the left upper limb, which consisted of internal rotation and adduction at the shoulder, extreme extension of the elbow, hyperpronation of the forearm, with slight flexion of the wrist and fingers. This "extension reflex" had a somewhat sudden onset after a latent period in response to appropriate stimulation.

The post-mortem examination revealed an area of softening, involving the posterior part of the right lenticular nucleus and the right internal capsule. There was general thickening of the pia mater, and sclerotic changes in the cerebral vessels. The spinal cord showed chronic lepto-meningitis involving the posterior surface with atrophy of the posterior columns and the posterior roots.

There were œdema and congestion of the lungs, chronic interstitial nephritis, atheroma of the aorta, and sclerotic changes of the cardiac valves.

REFERENCES.

[1] BABINSKI, "Du phénomène des orteils et de sa valeur sémiologique," *Semaine Médicale*, 1898, **18,** 321.

[2] BEEVOR, CHARLES. "The Croonian Lectures on Muscular Movements and their Representation in the Central Nervous System," London, 1904, 54.

[3] BROWN, GRAHAM. "On the Occurrence of a Plastic Flexor Tone in the Monkey," *Journ. of Physiol.*, 1915, **49,** 180,

[4] HEAD, HENRY, and RIDDOCH, GEORGE. "The Automatic Bladder, Excessive Sweating and some other Reflex Conditions in Gross Injuries of the Spinal Cord," *Brain*, 1917, **40,** 217.

[5] KELLING, GEORG. "Untersuchungen über die Spannungszustände der Bauchwand," *Zeitschr, f. Biol.*, 1903, Bd. **44,** S. 161.

[6] RIDDOCH, GEORGE. "The Reflex Functions of the Completely Divided Spinal Cord in Man," *Brain*, 1917, **40**, 264.

[7] *Idem.* "The Clinical Picture of Complete Transverse Division of the Spinal Cord," *Medical Science*, 1921, **5,** 49.

[8] SHERRINGTON, C. S. "The Integrative Action of the Nervous System," London, 1906.

[9] *Idem.* "Cataleptoid Reflexes in the Monkey," *Proc. Roy. Soc.*, 1897, **60,** 412.

[10] *Idem.* "Flexion-Reflex of the Limb, Crossed Extension Reflex and Reflex Stepping and Standing," *Journ. of Physiol.*, 1910, **40,** 105.

[11] *Idem.* "The Postural Activity of Muscle and Nerve," *Brain*, 1915, **38,** 212.

[12] *Idem.* "Decerebrate Rigidity and Reflex Co-ordination of Movements," *Journ. of Physiol.*, 1898, **22,** 319.

[13] *Idem.* "Reciprocal Innervation." Seventeenth International Congress of Medicine, London, 1913, Section II, Physiology, part i, 85.

[14] TILNEY and RILEY. "The Form and Functions of the Central Nervous System." London, 1921, 154.

[15] VIETS, HENRY. "Relation of the Form of the Knee-jerk and Patellar Clonus to Muscle Tonus," *Brain*, 1920, **43,** 269.

[16] WALSHE, F. M. R. "The Physiological Significance of the Reflex Phenomena in Spastic Paralysis of the Lower Limbs," *Brain*, 1914, **37,** 269.

[17] *Idem.* "On the Genesis and Physiological Significance of Spasticity and other Disorders of Motor Innervation," *Brain*, 1919, **42,** 16.

[18] WILSON, S. A. KINNIER.—"On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits," *Brain*, 1920, **43,** 220.

DYSSYNERGIA CEREBELLARIS MYOCLONICA—PRIMARY ATROPHY OF THE DENTATE SYSTEM: A CONTRIBUTION TO THE PATHOLOGY AND SYMPTOMATOLOGY OF THE CEREBELLUM.[1]

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CHAPTER I.—INTRODUCTION.

SOME years ago, under the title "Dyssynergia Cerebellaris Progressiva, or Chronic Progressive Cerebellar Tremor" [10] I directed attention to a peculiar disorder of motility, which I regarded as a definite clinical type of nervous disease.

This affection was characterized by generalized intention tremors, which began as a local manifestation and gradually extended to other parts of the voluntary muscular system. The extremities, and more especially the arms, showed the greatest degree of involvement.

The coarse ataxic-tremor disturbance which characterized the dis-

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order was only present when the muscles were in action, and ceased entirely during relaxation and rest. It was, therefore, volitional in character and of the "intention tremor type." Another peculiar feature was the severe involvement of the finer co-ordinated movements of the extremities, while the coarser and more massive movements underlying station and locomotion were but little affected.

When this tremor disturbance was subjected to careful analysis there was found associated with it a disorder of muscle tone and of the ability to measure direct and associate muscular movements, the clinical manifestations of which were dyssynergia, dysmetria, adiadokokinesis, hypotonia and asthenia. All of these symptoms, including the volitional tremor, which was an extreme expression of the underlying disturbance of muscle tone and synergy, showed the existence of a fundamental disorder of cerebellar function.

I, therefore, regarded the affection with its progressive tendency, chronic course and well-defined cerebellar symptomatology as "an organic disease caused by degeneration of certain special structures of the cerebellar mechanism, which were concerned in the regulation of the tonus and synergies of muscles."

Since my original publication, I have had occasion to observe another group of cases which combined the symptomatology of dyssynergia cerebellaris progressiva and myoclonus-epilepsy. There was the progressive dyssynergia characteristic of a cerebellar disorder, in association with epilepsy and myoclonus. Here, as in the group of cases uncomplicated by myoclonus-epilepsy, the movements of the extremities showed the greatest degree of disturbance.

This form of dyssynergia, which I would term dyssynergia cerebellaris myoclonica, I have also observed in association with Friedreich's ataxia, a combined cerebello-spinal involvement, which is not infrequent in the history of cerebellar system disease. In one of these cases careful pathological investigations were made which throw considerable light on the origin and anatomical basis of the cerebellar portion of the symptomatology. On the basis of these studies I would refer the progressive dyssynergia to a primary atrophy of the efferent dentate system of the cerebellum, and would regard this system as the essential neural mechanism underlying the production of the cerebellar or intention tremor.

In conclusion, I will state my views on the relation of the cerebellum to the static or posture system of motility, and the bearing which this conception has on the interpretation of cerebellar function.

Chapter II.—Dyssynergia Cerebellaris Progressiva Myoclonica: a Form of Progressive Cerebellar Tremor associated with Myoclonus-Epilepsy.

Critical Report of Four Cases.

Case 1.

Summary.—Myoclonus-epilepsy began at the age of 17, and was followed five years later by symptoms of dyssynergia cerebellaris progressiva, viz., generalized intention tremors and scanning speech, associated with dysmetria, dysdiadokokinesis, hypotonia and asthenia.

The patient, a man aged 30, single and a telegraphist by occupation, was referred to the Neurological Institute, in May, 1918, and was admitted to the service of Dr. Pearce Bailey. I am indebted to Dr. Bailey for the privilege of observing and reporting the case.

Family history.—His father was a periodical drinker and two of the father's uncles were alcoholic. An aunt on the father's side was subject to fainting attacks of rather doubtful nature and which were not called epileptic by the family. Their character could not be determined with certainty. His mother is living and in good health. Parents were not related. One sister is alive and well. A brother died in early life. No other member of the family, so far as he knows, has had any disease similar to the one from which he is suffering.

The previous history shows nothing unusual. Childhood was normal, he did good work at school and was quite up to the average in his mental development. He had measles and whooping-cough in early life. There was no history of trauma, venereal disease or of alcoholic excesses.

Onset.—The first symptoms of his disease appeared at the age of 17. At this time he had an epileptic seizure which was characterized by clonic muscular spasms of the right arm and leg, which developed while wrestling and lasted only a few minutes. He had two such attacks, neither of which were associated with complete unconsciousness but in both he became dizzy and fell to the ground. After these initial seizures the subsequent attacks were always accompanied by complete loss of consciousness. They were nearly always preceded by a motor aura, consisting of a progressive spasm of the right arm and leg, which terminated in unconsciousness, and was followed by a general convulsion. The tongue was often bitten, and there were occasional incontinence of urine and the other manifestations of a major epileptic seizure. These grand-mal attacks were comparatively infrequent, occurring only three or four times a year, and at one time there was a free interval of nearly three years.

When he was 19 years of age, that is, two years after the first epileptic seizure, the myoclonic jerks made their appearance. These were usually worse in the early morning hours and gradually increased in extent and intensity. He states that they were usually diminished by the use of alcohol, and the

patient sometimes took this means of relieving their severity. The myoclonus first appeared in the hands and arms and gradually extended to the trunk and lower extremities. Subsequently, the myoclonus became so severe that he was occasionally thrown to the ground and on one of these occasions he broke his nose.

Five years after the onset of the myoclonus-epilepsy the intention tremor made its appearance in the arms. This gradually increased in severity and later the speech became affected, and the legs showed evidences of involvement.

There has been no headache, no diplopia and he states no special impairment of memory. Preceding the epileptic attacks there is a whirring sensation in the head but otherwise no vertiginous seizures. The myoclonus is increased by mental stress and worry, and varies in its intensity from day to day. He has good days when the involuntary jerks and starts are in abeyance and other days when they are very severe.

Physical examination.—Shows a well-developed muscular man. The speech is slow and scanning in character and of the cerebellar dysarthric and explosive type. There is a well-marked, typical intention tremor of both upper extremities, similar in all respects to that observed in multiple sclerosis. In addition there is also a typical intention tremor of both lower extremities. Associated with the intention tremor are evidences of dysmetria and dysdiadokokinesis.

There is no loss of the gross motor power of arms or legs and no muscular atrophy. There is, however, in the arms, some diminution of the power to maintain fixed muscular contractions and postures except for short periods of time, due to tremor and asthenia. For example, when he extends the arm from the body in abduction, and attempts to hold it in this position, there ensues a coarse ataxic tremor and after a brief effort the arm is dropped to the side, in this respect simulating the behaviour of the intention tremor in multiple sclerosis. There is difficulty in maintaining a fixed posture of the upper extremities except for short periods of time. There is also a moderate degree of hypotonia in both upper and lower extremities. The Stewart-Holmes sign of cerebellar hypotonia is however not demonstrable.

The tendon reflexes of both upper and lower extremities are present, active and equal on the two sides. There is no ankle clonus, no patellar clonus and the plantar reflex is of the normal flexor type (no Babinski response). The cremasteric and abdominal reflexes are present and equal on the two sides. There is no history of pain or of paræsthesias. All sensations both superficial and deep are normal.

The pupils are equal and react to light and accommodation. The ocular excursions are normal and there is no nystagmus. Fundi negative. The innervation of the face, tongue, soft palate and muscles of mastication are also normal. Save for occasional myoclonic jerks there is no Rombergism and the station is normal. The spine is normal in contour and in movement.

During the course of the examination there are frequent myoclonic starts or jerks in both upper and lower extremities and trunk. This tendency is

increased or may be induced by voluntary movement, and this together with the intention tremor produces a very grave disorder of muscular co-ordination.

When lying on the back patient is relaxed and perfectly quiet, but any attempt at movement is likely to initiate myoclonic twitchings of the face, extremities or trunk. This tendency is increased by talking, or indeed by any form of volitional movement. In the recumbent position he can elevate both the upper and lower extremities, and hold them in this position with closed eyes without developing ataxic movements. The myoclonic jerks also occur spontaneously apparently without the intervention of volitional effort.

He is able to stand and walk, but does so with great caution and some uncertainty as if anticipating a myoclonic jerk. The intention tremors of the arms and legs occur with all volitional movements and present all the typical characteristics of this form of tremor. It is readily distinguished from myoclonic jerks although frequently accompanied by them. Associated with the intention tremor are typical dysmetria and dysdiadokokinesis. The gait and station, while showing some uncertainty, are not markedly titubating in character and the evidences of cerebellar disorder affect deeply the higher forms of motility, viz., speech and the volitional movements of the extremities (appendicular dyssynergia).

The heart and lungs are normal. Abdominal palpation is negative. There is no demonstrable enlargement of the hepatic area, and no evidences of abnormal pigmentation of the skin or cornea. Urine normal; Wassermann test of blood negative.

Comment.—This patient presents in addition to the symptoms of myoclonus-epilepsy a chronic progressive dyssynergia of cerebellar origin. With the scanning speech and intention tremors of both upper and lower extremities there are the associated manifestations of cerebellar disease, viz., dysmetria dysdiadokokinesis, hypotonia and asthenia. With the exception of epilepsy, myoclonus and dyssynergia there are no other evidences of organic disease of the nervous system. It is of interest to note that the evidences of dyssynergia are appendicular rather than trunkal in distribution and that higher types of movement are chiefly affected. There was no familial history of either myoclonus-epilepsy or cerebellar disease.

Case 2.

Summary.—A girl, aged 19; onset of myoclonus-epilepsy, at the age of 7. Associated with this is a progressive dyssynergia of cerebellar origin, affecting more especially the speech and extremities.

This patient is 19 years of age, and single. She was admitted to the Craig Colony for Epileptics on January 14, 1918, and is still under observation in that institution, where I saw her in consultation with Dr. Doolittle of the Colony Staff.

Family history.—Father has been excessive in the use of alcohol. Her mother is in good health. Parents are not related. She has three brothers

and two sisters, all in good health. There is no family history of any similar nervous disorder.

Personal history.—The patient was born at full term following a labour lasting forty-eight hours. The delivery was said to have been natural. Among possible pre-natal influences may be mentioned her father's alcoholism and mother's worry over family affairs. There was no paralysis or spasm immediately after birth. Her mind developed naturally during infancy and childhood, and she was considered very bright in school. She had whooping-cough at 10 years, measles at 13 years, and diphtheria at 14.

Present illness.—At the age of 7 it was first noticed that her hands would shake and jerk while feeding herself or trying to pick up things. This tendency to myoclonus continued and gradually increased in extent and severity, finally involving, in addition to the arms, the legs, trunk, and even the facial musculature.

Three years later, when she was 10 years old, she had her first epileptic seizure. These attacks recurred every three or four months. They were diurnal and of the grand-mal type. At the present time she averages about six grand-mal seizures a year. They are of typical character, and are frequently associated with biting of the tongue and incontinence of urine. It was difficult to obtain any very definite statement as to the time of the appearance of the intention tremor, because of the existence and severe nature of the myoclonic phenomena which tended to mask the other motor disorder.

For the last five years, however, a coarse ataxic tremor of the arms and legs has been present on volitional movements and the speech has been jerky, irregular, explosive. These symptoms have grown gradually worse. It is more than probable that the characteristic symptoms of a cerebellar disorder were present before the fourteenth year, and were overshadowed by the myoclonia.

Physical examination (February, 1918).—During the course of the examination, while patient is seated in a chair, there are frequent sudden myoclonic jerks or starts, which may appear spontaneously, but usually precede or follow some volitional effort. These may appear in the arms, trunk, legs, or face; as a rule, there is a single coarse jerk or start, and then an interval—occasionally a small series of myoclonic starts occur in rapid succession. These movements are brusque, sudden, and involve synergic groups of muscles and produce a considerable locomotor effect. On arising from a sitting posture there is at first a slight unsteadiness and an effort to restore the equilibrium by standing upon a broad base with balancing. While walking, the harmony of movement is frequently broken by coarse myoclonus jerks which throw her to one side, causing a loss of balance.

These coarse myoclonus jerks appear in the trunk and arms and legs. They also occur while sitting quietly in a chair, producing sudden brusque jerks of the trunk or extremities. A slight flicker of myoclonic character is occasionally observed in the face. The myoclonus is very apt to occur just before, apparently initiated by the response to a voluntary movement, such as lifting up the arm, placing finger on the tip of the nose, or a movement of a

leg. At certain times, and on certain days, they are much more severe than at others, and in the morning they occasionally cause her to toss about in the bed so that she almost falls out. This patient's myoclonic movements sometimes continue during sleep and disturb her rest to a considerable extent. The abdominal muscles are also occasionally the seat of spasm. The myoclonic movements of the extremities are often symmetrical, both sides being simultaneously involved. The difficulties of gait and station are very much increased by the tendency to myoclonus, as all of the usual muscular activities of standing and walking in themselves produce myoclonus, and are the cause of constant uncertainty and inco-ordination.

She is able to stand alone, and there is no Rombergism. She walks on a rather broad base, lifting the foot too high with jerky, irregular ataxic-like movements of the legs (dysmetria). The attitude of the trunk is also somewhat oscillating, showing a slight tendency to balancing. The spine is straight, and there is no scoliosis.

A cerebellar or intention tremor is present in both upper extremities, more particularly on the left side. It is of the same character as the intention tremor of multiple sclerosis and is brought out conspicuously by the finger-nose test. On the performance of this test there is the coarse atactiform shaking which increases in intensity as the finger approaches the tip of the nose. And there is the quick jerking away movement of the fingers from the tip of the nose, as in true intention tremor. Well marked adiadokokinesis and evidences of dysmetria are also present on both sides.

In the recumbent posture there is also dysmetria in the heel-knee test, and volitional movements are irregular, jerky and of the cerebellar ataxic type. The movements during these tests are frequently interrupted by myoclonic jerks. There is no noteworthy hypotonia of the extremities and the Stewart-Holmes sign of hypotonia is absent.

The tendon reflexes of the arms (supinator, biceps and triceps jerks) are present and not exaggerated. The knee-jerks are active and equal on the two sides. Both Achilles jerks are present and equal. There is no clonus. The abdominal reflexes are present and equal. The plantar reflexes are also present and of the normal flexor type (no Babinski).

The pupillary reflexes are normal. The ocular excursions are normal and there is no nystagmus. Fundi normal. The innervation of the face, tongue, soft palate and muscles of mastication are normal.

The speech shows well-marked dysarthria of the cerebellar type. It is jerky, explosive and at times scanning.

There is no Romberg symptom and no ataxia of station develops on closure of the eyes. In the recumbent posture both the upper and lower extremities may be elevated and held in this position with closed eyes without the development of ataxic movements.

Sensation.—The superficial sensibility is normal. The sense of touch, pain and temperature are well preserved. The deep sensibility is also normal and there is no disorder of the sense of posture.

There is no disturbance of the sphincter control of the bladder or rectum.

The intelligence shows some general reduction and retardation. There is loss of memory and slowness of thought and action. Judgment is poor. In the Binet-Simon test she is rated at 9 years.

The heart and lungs are normal. The urine is normal. Wassermann test of the blood is negative. There are no evidences of abnormal pigmentary deposits on the skin or at the sclero-corneal junction.

Comment.—With the exception of myoclonus-epilepsy and progressive dyssynergia there are no other evidences of organic disease of the nervous system. The chief disorder of cerebellar function is that affecting articulation and the movements of the extremities. The gait and general equilibrium also show some disorder of cerebellar function, which is accentuated by the severe type of myoclonia which is present.

In this case the dyssynergia while preponderantly appendicular in distribution also involves to some extent the trunkal musculature. As in the previous case, there was no history of the familial occurrence of either myoclonus-epilepsy or cerebellar disease.

CASE 3.

Summary.—Myoclonus-epilepsy of twenty-six years' duration. Associated with symptoms of dyssynergia cerebellaris progressiva, viz., cerebellar dysarthria, intention tremor, dysmetria and adiadokokinesis, affecting chiefly the volitional movements of the extremities.

This patient was a man, aged 38, single, an inmate of Craig Colony for Epileptics, where I saw him in consultation with Dr. Shaw of the Colony staff.

Family history.—Parents are living and in good health, patient is an only child of his fraternity. He has one half sister (mother's daughter by first marriage), aged 42, who is married and in good health. Careful inquiry by letters and interviews failed to reveal any history of epilepsy or other nervous disorder among the relatives.

Personal history.—The history of the birth, of infancy and of childhood were negative up to the age of 12 years. He had whooping-cough at 8, and measles at 9. There were no untoward symptoms during these infections, which would indicate involvement of the central nervous system. School progress was about the average, and at the age of 16 he left school to work on a farm. He continued this occupation up to the time of his admission to the Colony in the summer of 1912. His habits were moderate, he used tobacco in moderation and beer occasionally. There was no history of injury or of venereal infection.

History of the disease.—Epilepsy: his first seizure occurred at the age of 12 years. He was on his way from school, stopped to tie his shoe-string, and fell over in a convulsion. "Rush of blood to the head" was the assigned cause. Soon after this, myoclonic jerks and twitchings made their appearance. These gradually increased in severity and frequency and were especially

severe in the mornings. Myoclonia was usually greatly exaggerated before convulsive phenomena.

His general nervousness and the severe myoclonia were the reasons for his seeking admission to the Craig Colony in 1912.

Upon admission he was in good general health, and weighed 187 lb. He was "nervous and unstable." There were myoclonic jerks involving the head, trunk, upper and lower extremities.

His general intelligence was fair.

During the six years of his Colony residence, he has slightly deteriorated mentally, and has lost weight, but not rapidly. The myoclonia has greatly increased. The speech defect which was slight on admission has become intensified. There are exhaustion states following severe attacks of myoclonia, which at times may exist for days, and are quite general in their distribution.

It is definitely established that during the early years of his disorder he had severe grand-mal seizures, the tongue was bitten and there are scars about the face and scalp due to injuries received by falling during attacks. From 1912 to 1916 there are recorded occasional seizures, which appear on the regular Colony records, but most careful investigation fails to reveal, even from those in daily contact with him, that he has had attacks other than myoclonia in recent years. The present nurse in charge of his cottage, who has observed him for the greater part of five years, denies the occurrence of seizures, and states that the only manifestations of his illness are the "jerks and tremors." It is quite certain that the years of 1916, 1917 and 1918 have found him free from epileptic seizures, as he has been under close observation during that time. He has averaged during this period about two weeks per year in bed as a result of myoclonia, which made it impossible for him to get about, or even to feed himself.

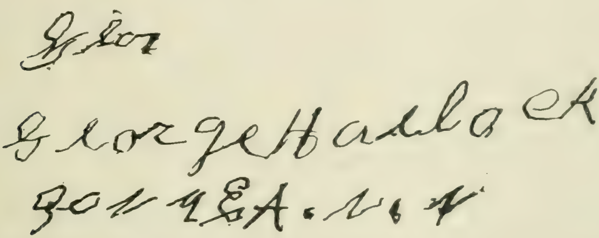
Physical examination. — Patient is a muscular, well-developed man. Station is steady and there is no Romberg symptom on closing the eyes. The spine is normal in contour and movements. The gait shows a slight clumsiness and awkwardness, especially on turning corners or facing about suddenly. At these times there is a slight tendency to uncertainty and the smooth harmony of balance and locomotion is somewhat disturbed. There is, however, no definite or distinct tendency to titubation and the slight disorder of gait and station might be regarded as merely clumsiness were it not for the existence of other and more characteristic cerebellar symptoms in the more delicate movements of the extremities.

The pupils are equal and react to light and accommodation. The ocular excursions are normal and there is no nystagmus (no history of diplopia). The other cranial nerves are negative. Fundi are normal.

Speech is of the cerebellar type, slow, of scanning character, and at times explosive. The tongue is protruded in the median line, without tremor. The innervation of the face, masseters and soft palate are normal. Frequently during the examination coarse myoclonic jerks and jumps occur in the extremities and trunk. These may be spontaneous but are particularly likely to

be initiated by any volitional effort or movement. At times the myoclonus occurs immediately after the cessation of a movement. Occasionally it is symmetrical on the two sides of the body, but both in the arms and legs independent myoclonic contractions are common. As in other cases the face is also occasionally involved.

There is no diminution of the gross motor power of the extremities and no evidences of muscular atrophy. On attempting to place the index finger on the tip of the nose a well-marked intention tremor is developed, which is more pronounced on the left side. This varies somewhat in intensity. At times there is present the coarse, slow, ataxic movement of dyssynergia which increases as the finger approaches the nose, on other occasions it has more the character of the typical intention tremor of multiple sclerosis. Evidences of dysmetria and typical adiadokokinesis are also present in both upper extremities, more particularly on the left side.



Patient's signature July 23, 1918, during "quiescent period" of the myoclonia.

In the lower extremities, evidences of cerebellar ataxia (dyssynergia and dysmetria) are also elicited when volitional movements are attempted, such as placing the heel upon the knee. There is no definite hypotonicity of the muscles of either arms or legs, and the Stewart-Holmes sign of hypotonia is absent.

Reflexes.—The arm jerks (supinator, biceps and triceps jerks) are present and active. The knee jerks and Achilles jerks are also present and active. There is no clonus. The skin reflexes are normal. The abdominal and cremasteric reflexes are elicited and the plantar reflexes are present and of the flexor type (no Babinski).

All sensations, both superficial and deep, are normal. The sense of position is quite normal in both upper and lower extremities. Abdominal palpation is negative and there is no enlargement of the hepatic areas, no abnormal pigmentary deposit of skin and sclera. The heart and lungs are normal. Urine is normal. Wassermann test of the blood is normal.

Comment.—In this patient, as in the others, there was difficulty in determining the approximate age of onset of the dyssynergia. The myoclonus-epilepsy is itself such a formidable condition and causes so severe a disturbance

of motility that it rather tends to mask the other disorder. With the exception of myoclonus-epilepsy and dyssynergia no other symptoms of organic disease of the central nervous system could be found. Symptoms indicative of multiple sclerosis, Friedreich's ataxia or gross cerebellar disease were not present. I would therefore regard the condition as one of dyssynergia cerebellaris associated with myoclonus-epilepsy.

There was no familial history of either disorder.

Case 4.

Summary.—A girl 15 years of age with nocturnal epilepsy, myoclonia and slight cerebellar dyssynergia of three years duration.

A young girl, 15 years of age, a private patient, first came under my care in July, 1921. The family history, so far as could be elicited, was negative. The father, a professional man, died as the result of an accident. Up to the time of his death, at the age of 45, he was in good health and had never presented any symptoms of nervous or mental disease. Mother is living and well. There is no history of epilepsy, myoclonia or other organic nervous disease in their forebears. The parents were not related.

Previous history.—Patient is an only child, born at term, normal pregnancy and non-instrumental delivery. She was breast fed for a year and with the exception of digestive disturbances in early life was a normal infant. No infantile convulsions. Menses began at the age of 12, and are quite irregular. She was sent to school at the usual age and is of average intelligence.

History of present illness dates back about three years and began with what have been called nightmares. She awakens in the night with a scream and passes into general convulsive attack with unconsciousness, frothing at the mouth, lasting several minutes, and followed by a period of mental confusion. At first these attacks were infrequent but later they increased in number and severity and now average once a week, sometimes oftener. Two years ago, that is, one year after the beginning of the nocturnal epileptic seizures, curious muscular jerks or twitchings made their appearance. At first these were not severe and were more marked on certain days, usually the day before the nocturnal seizures. The arms, legs and trunks were variously affected, later speech became involved and there was a slight tendency to myoclonic waves and twitching of the face. This tendency to myoclonus has gradually grown worse and on bad days it is rather difficult for her to walk alone and use her arms in dressing and eating—on these occasions she usually remains in bed.

On these bad days she sometimes falls to the ground while walking or standing, the legs suddenly giving way under her. On two occasions she had severe falls of this character, apparently associated with momentary blurring of consciousness. The patient insists, however, that in these falls there is no vertiginous sensation or momentary lapse of consciousness and she simply suddenly loses the ability to stand upright, her legs giving way under her. On one occasion when I was testing her gait and equilibrium she plunged quite

suddenly to the floor, all of a heap, striking her chin rather heavily. She immediately rose to her feet and was a little shaken from the sudden and severe fall, but as far as I could determine there was no obscuration of consciousness or pallor, as one observes in petit mal. The attack was caused by a sudden release of the muscular mechanism which underlies the posture of standing and is probably a massive expression of the myoclonia. In one of these she suffered quite a severe laceration of the scalp. As a rule the myoclonia is most severe a few hours before an attack and it is usually possible to foretell the epileptic seizures by this aggravation of the myoclonic phenomena.

A nurse who has slept in the room with the patient for a number of months gives the following description of the seizure. "An attack, or nightmare, is usually preceded by an unsteady afternoon and evening: a difficulty in eating and talking and an inability to walk unaided is shown at these times. The nightmare usually begins with a series of screams and guttural cries followed by a clenching of the teeth and tautening of all the muscles of the body. It is an impossibility to even move one of her fingers at this time. After an interval of ten minutes, or sometimes longer, there occur great convulsions of the body followed by profuse foaming at the mouth. If attempts to awaken the girl are successful she invariably seems dazed and very often lapses into childish manner of speech—sometimes half weeping—babbling some nonsense—repeating some word or sentence. Twice she has played with her toes and wept when I forced her to stop. Sometimes it takes more than half an hour to persuade her to lie down and sleep after a seizure. The day following she invariably has a dull nervous headache which lessens as the day goes on. The second day following is usually her strongest, brightest day. Nightmares occur on the average of once a week, but sometimes there are three, and rarely more than one in a night."

In addition to the nocturnal crises and the myoclonus, another group of symptoms has been observed, viz., a certain awkwardness or inco-ordination of movement. It is difficult to place the exact time of the appearance of these symptoms, for the reason that the night crises and myoclonia have dominated the clinical picture. It is quite certain however that they have been in existence for the two years, gradually growing more severe. (The mother states that even before the appearance of the nocturnal seizures the movements of the extremities were somewhat clumsy and awkward, and this was especially noticeable in her table manners. This clumsiness was not taken seriously at the time and was regarded simply as a peculiarity of childhood, which would pass off in the course of time.) There is a certain awkwardness in walking and a tendency to balancing on rising from a sitting posture. There is an awkwardness and uncertainty in the use of the arms and hands and a thickness and uncertainty of articulation.

Mentally she is bright and intelligent, but memory is not so good as formerly, and she is at times peevish and irritable, especially on the bad days

when the myoclonia is exaggerated. She is subject to headaches after the nocturnal crises, but not at other times, and is in good general health.

Physical examination.—She is well developed, of average height and muscular development. Seated in a chair and while conversing certain of the muscles of the face, trunk or arms will exhibit a quick muscular contraction, producing a marked locomotor effect. During the interval between these moments of myoclonia her expression and posture are perfectly normal. The myoclonic jerks are frequently initiated by a voluntary movement, such as reaching for some object, an effort to speak or a movement of the head. Usually there is a single jump or jerk and then rest. Occasionally a series of such movements occur and the trunk or limbs are thrown about in a rather bizarre manner.

The pupils are equal and react to light and accommodation. There is no nystagmus. The optic nerves are normal. All of the motor cranial nerves are normal. The articulation is slow, jerky and uncertain. In addition to occasional myoclonic speech disturbance there is slight dysarthria of cerebellar quality.

The upper extremities are normal in their gross motor power and there is no tremor of the outstretched hands. There is however a slight ataxia on the finger-nose test, and a distinct tendency to dysmetria and a very typical dysdiadokokinesis of both arms and fingers. There is also slight hypotonia of the musculature. The Stewart-Holmes sign of hypotonia is absent.

On arising from a sitting posture there is a certain static ataxia and a tendency to balance the trunk. In walking the legs are spread somewhat and there is a moderate degree of cerebellar disorder of progression which is especially obvious on turning corners or turning round suddenly.

In the recumbent posture the heel-knee test shows a moderate degree of dysmetria. There is however no obvious hypotonia. On elevating both arms and legs in the recumbent posture, with the eyes closed, there is no ataxia.

The tendon reflexes of both upper and lower extremities are present and equal on the two sides, viz., the supinator, biceps, triceps, knee jerks and Achilles jerks. Sensation, both superficial and deep, is normal. The spinal column is of normal contour and there is no deformity of the feet. The heart and lungs are normal. Abdomen is normal on palpation and percussion. There is no enlargement of the liver. There is no pigmentation of the skin or cornea. Urine shows no abnormality.

Comment.—The case is of interest as representing a somewhat earlier stage of the dyssynergia cerebellaris myoclonica. Here again there is no family history of myoclonus-epilepsy or of a cerebellar disorder.

Remarks.—The association of cerebellar dyssynergia and myoclonus-epilepsy in this group of cases suggests the occurrence of two independent nervous disorders in one individual. Combined forms of the various congenital, familial and hereditary types of nervous disease are

by no means uncommon, and these associations are especially frequent in the spinal and cerebellar groups of system diseases. For example, Jendrassik [14], who has devoted particular attention to this subject, records many such combinations. He mentions a combined form of Friedreich's ataxia and muscular dystrophy, of spino-cerebellar ataxia and muscular dystrophy, and of Friedreich's ataxia with Huntington's chorea. He also cites the occurrence of myoclonus in association with optic atrophy. André-Thomas [3] has also described a combined form of olivo-ponto-cerebellar atrophy and spinal ataxia.

Such combinations, however, are rare, and show merely a predisposition to the two disorders in the same individual, and would not necessarily indicate any essential relationship between them.

On the other hand, little is known at the present time of the pathology and localization of myoclonus. Its occurrence, therefore, in conjunction with a special type of cerebellar disease, is not without interest, and may have a deeper pathological significance than would appear.

Sioli [19], in a histological study of one of Rechtenwald's cases of myoclonus-epilepsy, found rather widely disseminated histological changes.

In the cerebellum there were enormous collections of lipoid material in the region of the corpus dentatum in the white matter of the cerebellar hemispheres and in the pons. There was also a considerable increase in the number of glia cells. The cells of the corpus dentatum were normal. In the cerebral cortex there was also an excessive deposit of lipoid material in the ganglion cells and glia. The glia cells and fibres were increased in the marginal layer. The cortical changes were all more conspicuous in the region of the central convolutions. The only alteration noted in the spinal cord was atrophy of Hellweg's triangular bundle (olivo-spinal tract).

In the present state of our knowledge, while these two disorders may be regarded as independent manifestations of two distinct disorders in the same individual, the possibility of a closer pathological association should be borne in mind, and the cerebellar mechanism should be carefully considered, both clinically and pathologically, in all cases of so-called myoclonus-epilepsy.

Chapter III.—Dyssynergia Cerebellaris Myoclonica, associated with Friedreich's Ataxia.

Clinical Report of Two Cases, One with Histological Study of the Central Nervous System.

In the previous chapter there was described a group of cases, characterized by progressive dyssynergy, associated with myoclonus-epilepsy. In none of these cases was there any history of an hereditary or familial tendency to either disorder.

In the present chapter I shall present another group of cases in which myoclonus-epilepsy and dyssynergia cerebellaris progressiva were associated with symptoms of Friedreich's ataxia. It is well known that the various cerebellar atrophies and degenerations are not infrequently associated with evidence of spinal cord involvement. Friedreich's hereditary spinal ataxia and Marie's so-called hereditary cerebellar ataxia are classical examples of this association, and may exist alone or in combination. The progressive cerebellar dyssynergia is apparently no exception to this rule—it may exist alone or in combination with spinal cord involvement of the Friedreich's type.

Case 5.

Summary.—A man, aged 36, with symptoms of myoclonus-epilepsy since his twenty-first year. Previous to this there were symptoms of Friedreich's ataxia and progressive cerebellar tremor, which steadily progressed. Pathological study shows the typical spinal lesions of Friedreich's ataxia associated with a primary atrophy of the efferent dentate system of the cerebellum, viz.: considerable diminution in number and extensive atrophy of the cells of the dentate nucleus, with secondary atrophic changes in the superior cerebellar peduncles.

The patient, a man, aged 36, single, was admitted to my service at the Montefiore Home and Hospital on April 15, 1918.

Family history.—His father is living and well. Mother died at 51 from heart disease. The parents were not related. One brother died at age of 2 from pneumonia. He has two other brothers and one sister living and in good health. There is no history of tuberculosis, diabetes, epilepsy, or other nervous disease in the family, with the exception of a twin brother who is suffering from the same malady, and whose case will be presently described.

Previous history.—Had measles in childhood. Was always moderate in the use of alcohol, coffee, and tea. Smoked cigarettes in moderation. Denies venereal infection. No history of trauma.

Present illness.—Began fifteen years ago with attacks of unconsciousness

and convulsive seizures. Patient believes these attacks were caused by fright following a burn. At the time of the accident he was incapacitated for some weeks. The epileptic seizures increased in frequency, and he sometimes fainted on the street and was carried home. A few months later there appeared coarse jerky movements of the extremities. The myoclonus jerks or starts came on gradually and steadily increased in severity and extent.

Even in the earlier juvenile period it was noticed that his manner of walking was awkward and uncertain, and this steadily increased. For the past ten years walking has been unsteady and difficult, speech has been slow and there has also been some uncertainty in the use of the hands. He thinks that part of his inability to walk is due to the fear of falling on account of the myoclonic jerks and starts, which develop on making the slightest effort.

For the past seven years his speech has been quite indistinct and for about the same period of time he has had a marked volitional tremor of the hands on attempting to grasp an object. For three or four years he has suffered from very extreme weakness and unsteadiness of lower extremities and is unable to walk.

During the whole course of the disease there have been no subjective sensory disorders, such as pains or paræsthesias, and there has been no difficulty with the control of the sphincters of the rectum or bladder. States that he has not suffered from headache. He now wears dark glasses constantly, because any bright light increases the tendency to myoclonia. Hearing is not impaired. There are slight evidences of mental impairment and deterioration. His memory is poor, and his mental processes slow. He is querulous, irritable and easily excited.

Physical examination.—Patient is confined to a wheel chair, and is unable to stand or walk without assistance. He wears dark glasses and always seeks a quiet corner of the ward where there is no noise and no excitement. The slightest muscular effort brings on a series of severe myoclonic jerks or spasms which throw him brusquely to and fro in his chair or bed. Talking has the some effect as have also noises or any excitement. As a result of these continuous motor shocks the patient shuns conversation and any social contact. He wishes to be left alone and in quiet.

His speech is slow and typically scanning, and at times uneven and explosive. It is often interrupted by myoclonic jerks and starts in the trunk and extremities, causing sudden shock-like movement. His head is frequently thrown backwards in sharp retro-colic clonus, associated with quick flexor movements of extremities. This larger myoclonic phenomenon is frequently repeated during the course of the examination. Occasional facial grimaces of myoclonic type are also noted. These movements are all accentuated by speech and voluntary movement. The clonic movements are said to occasionally persist in sleep, although in much diminished form. There is a typical and very marked intention tremor of both upper extremities which is associated with dysmetria and dysdiadokokinesis. On attempting to carry the finger tip to the nose the hand is thrown into a violent ataxic tremor similar to

that observed in an advanced case of multiple sclerosis. All tremor movement of the arm immediately ceases in a relaxed or recumbent posture. There are, however, occasional myoclonic starts at rest, which are increased by some one entering the examination-room suddenly. The musculature of the arms is hypotonic and the Stewart-Holmes sign of hypotonia is present in both upper extremities.

Asthenia is present in both arms, viz., when the attempt is made to extend the arm from the body in abduction and maintain the abductive extremity in this position. It can be maintained for a very brief period only, the arm falling to the side, a symptom which may be observed in the intention tremor of multiple sclerosis (asthenia or astasia).

The arm jerks are difficult to elicit and the supinator, biceps and triceps jerks are not definitely present. The abdominal reflexes are not elicitable. The cremasteric reflexes are present on both sides. The plantar reflexes are present but diminished and of the flexor type (no Babinski).

The movements of the lower extremities are extremely ataxic, but in addition to this there is on performing the heel-knee test a very marked intention tremor of both lower extremities. The legs are markedly hypotonic. The knee jerks are absent and the Achilles jerks cannot be elicited. He can stand and walk only with assistance. The Romberg symptom, owing to the extreme ataxia, cannot be tested. In walking, the legs are lifted high and thrown outward in a very ataxic and uncertain manner and resemble the gait of advanced locomotor ataxia. There is no definite spinal curvature but a tendency to pes cavus is present. There is general emaciation and wasting of the musculature but no localized degenerative atrophies.

Sensation.—There is a marked disturbance of the deep sensibility of the lower extremities, and loss of the sense of position. The deep sensibility of the hands is also affected and there is a loss of the sense of position in the hands and fingers. The superficial sensibility to touch, pain and temperature is diminished over the more distal portion of the lower extremities, but is nowhere abolished. The superficial sensation of the upper extremities is fairly well preserved: the only change being diminished acuity of perception over their distal portion. Myotatic irritability of the muscles is active and somewhat increased.

The pupils are equal and react to light and accommodation. Ocular excursions are normal. There is no nystagmus. Hearing is normal. The tongue is protruded in the median line; no atrophy and no tremor. Innervation of the palate is normal. The pharyngeal reflex is active. Facial innervation is normal as well as that of the muscles of mastication. Jaw jerk is not elicitable. The ophthalmological examination by Dr. Tenner revealed no abnormalities. The fundi were negative.

The heart and lungs are negative. Systolic blood pressure 110, diastolic 70. Wassermann test of blood negative. There are no pigmentary deposits of the skin or sclero-corneal margin.

Since his admission to the hospital patient has had very active myoclonia,

The head is thrown suddenly backward, the face is convulsed with occasional myoclonic waves, the arms flex and jerk, the trunk is thrown forward and the legs are flexed and tossed about in a wild and random manner. There are one or two discharges and then a period of rest, although these rest intervals are of very short duration. It is necessary to bind patient with a sheet to the chair, otherwise he would be thrown out, and even with this precaution the wheel-chair rolls around in a wild and reckless manner. This condition is very much aggravated by removal of the dark glasses and the excitement of any examination. Any visit or conversation induces an extreme degree of myoclonia. This condition gradually grew worse, the patient dying of exhaustion a few weeks later.

Comments.—This case differs from those described in the previous chapter in two respects, viz.: the association with Friedreich's ataxia and the familial incidence, a twin brother suffering from the same disease. The first symptoms of the cerebello-spinal disorder made their appearance in the early juvenile period. The myoclonus-epilepsy did not become manifest until the twenty-first year. The presence of the spinal symptoms of Friedreich's ataxia would mask many of the typical symptoms of cerebellar disease. The existence, however, of marked intention tremors and the scanning speech show very clearly the existence of cerebellar involvement (cerebellar tremor) and this was amply confirmed by histological examination.

The central nervous system was subjected to careful histological examination, and revealed the typical spinal lesion of Friedreich's ataxia, together with primary atrophy of the efferent dentate system of the cerebellum, viz.: diminution in number and atrophy of the dentate nucleus with atrophy of the superior cerebellar peduncles. (See report of histological examination.)

Case 6.

Summary.—A man, aged 29 years, the twin-brother of Case 5. Symptoms of dyssynergia cerebellaris and Friedreich's ataxia for the past ten years, gradually increasing in severity. Myoclonia made its appearance five years ago and epilepsy is only of three years duration.

History.—Patient is 29 years of age, the twin-brother of the case just described. He assists his father in business, who is a tailor by occupation. He resembles very closely his twin-brother in face and stature, and states that he has had symptoms of the same disorder for the past ten years or more. Before that he could run and do anything that other boys were able to do. The condition has been very slowly progressive.

It is characterized by thickness of speech, ataxia and intention tremor of the hands and arms, a certain awkwardness of the movements of walking and running and the symptoms of myoclonus epilepsy. At times there is a lateral tremor movement of the head.

For the past five years he has been subject to nervous shocks or starts, which at first occurred only three or four times a day, but have steadily increased in frequency and intensity. These attacks of myoclonus may

occur spontaneously but more frequently accompany attempts at voluntary movement. They have never approximated in severity those occurring in his brother's case. Efforts to talk are frequently accompanied by clonic "starts" and "jumps" (myoclonus), usually only a single movement which may involve the arms, face, neck, trunk and sometimes the legs.

Three years ago he had his first epileptic seizure. At first these attacks were infrequent but have gradually increased in frequency and during the past year have occurred as often as twice a week. In the seizures he is momenttarily unconscious and falls to the ground. The obscuration of consciousness is, however, of very short duration. It is as if his legs give way under him, and a darkness comes over his eyes, but he gets up immediately and goes on with what he was doing. There is no biting of the tongue, vomiting, or incontinence of the sphincters, and so far as could be learned no convulsive manifestations. This is a type of seizure which I have referred to the static or posture system of motility—static epilepsy. It is characterized by a sudden loss of control of the static or posture mechanism of standing with transitory obscuration of consciousness and without the usual convulsive manifestations of epilepsy. During the past few years his memory is not so good. There is no history of headache or diplopia, and the special senses are unimpaired. There is no vertigo; no tinnitus aurium; no attacks of nausea or vomiting.

Physical examination (April 28, 1918).—He is of medium height, slenderly built, and bears a very strong resemblance to his twin-brother. The pupils are equal and respond promptly to light and accommodation. The ocular excursions are normal and there is no nystagmus. Motor innervations of the face, tongue, soft palate and muscles of mastication are normal. The fundi are negative. At times, in certain positions a lateral tremor of the head develops.

The speech is dysarthric; rather slow and scanning and at times of a slightly explosive quality. The tongue is protruded in the median line; there is no atrophy or tremor and the palatal innervation is normal. The jaw jerk is not elicitable. Efforts to speak are often accompanied by mild clonic starts or jumps, usually, only a single one, which may involve the face, neck, arms, trunk and sometimes the legs. The station shows a slight unsteadiness but there is no special increase in this slight disorder of equilibrium on closure of the eyes. There is slight awkwardness in walking, especially on coming to a sudden stop or on turning corners. There is a moderate scoliosis in the mid-dorsal region, with a slight convexity towards the left. There is no muscular atrophy of the trunk, arms or legs and no fibrillary twitching. There is no deformity of the feet (no pes cavus). The gross motor power of the arms is normal. The movements, however, are awkward and ataxic and on attempting to carry out a volitional movement a typical intention tremor develops, which is more marked on the left side. The musculature of the upper extremities is moderately hypotonic and the Stewart-Holmes sign of hypotonia is present. (Handwriting, p. 509.)

The arm jerks cannot be elicited (supinator, biceps or triceps jerks). There is typical adiadokokinesis of both upper extremities. There is no loss of

gross motor power in the lower extremities and no evidence of muscular atrophy. There is a moderate degree of hypotonicity of the musculature and the knee jerks and Achilles jerks are absent. The plantar reflex is of the flexion type (no Babinski). The other skin reflexes, abdominal and cremasteric, are present and equal on the two sides.

On attempting to carry out the heel-knee test there are distinct evidences of dysmetria and of an ataxic disturbance, suggestive of the intention tremor but not so distinct as in the upper extremity.

Sensation appears to be normal. The superficial sensibility to touch, pain and temperature is intact, and the deep sensibility of the toes is well preserved. The muscle pain sense on pressure is well preserved.

Comment.—This case, like that of his brother, presents symptoms of dyssynergia cerebellaris progressiva, myoclonus-epilepsy and Friedreich's ataxia in combination.

According to the history, symptoms of cerebellar tremor and Friedreich's ataxia made their appearance some years after the development of myoclonus or epileptiform seizures. It is interesting to note that the epileptiform seizures correspond to what I have termed the static type of epilepsy, viz., a sudden loss of postural or static control without convulsive manifestations.

That the disorder in which we are interested has also been encountered by other observers is shown by the following extract from a case reported by Boschi [5] as ataxie héréditaire; paramyoclonus multiplex, type Unverricht.

The patient was a man 46 years old, who at the age of 15 was subject to convulsive seizures and myoclonus, associated with a well marked ataxic disorder, which had gradually shown a tendency to progression. There was in addition a very marked familial predisposition to the disease. The grandfather, father, and four brothers were all sufferers from the same malady.

Physical examination shows an ataxia of gait, of the movements of the trunk and of the arms. In both upper extremities there is present an intention

tremor. The speech is slow and dysarthric (bradylalia). The knee jerks are exaggerated and there is clonus. The pupillary reflexes are present but slow. The optic nerves are normal. There is a moderate mental defect and typical myoclonus of the Unverricht type.

The case, which is very briefly recorded, would seem to resemble Cases 5 and 6 of my series: dyssynergia cerebellaris myoclonica with Friedreich's ataxia. It is interesting because of the striking hereditary nature of the malady, which was absent in my series of cases.

Undoubtedly other cases have been observed and recorded, but, with this single exception, I have encountered in the literature no other examples in which myoclonus-epilepsy was observed in conjunction with evidence of a cerebello-spinal disorder.

Pathological Study of Case 5.

Autopsy Report.

The autopsy was performed thirty-eight hours after death, by Dr. J. H. Globus, Pathologist to the Montefiore Home and Hospital. His report is as follows :—

The body was that of a poorly developed male, 160 cm. in length. Skin thin, dry, pale, with several decubitus ulcers over lower sacrum, under the anterior superior spine of ilium and over internal tuberosity of right tibia.

The regional lymph nodes were normal in size and slightly palpable.

External genitalia were undeveloped, otherwise normal.

Scalp was free of hair and mottled with numerous small superficial ulcerations. Rigor mortis had disappeared.

On incising abdominal wall, a very small quantity of subcutaneous fat was found. On opening peritoneal cavity no adhesions; no fluid encountered.

Abdominal viscera were found in normal relation to each other and in normal position. No fluid and no adhesions in thoracic cavity.

Lungs, rather voluminous and collapsed only to slight degree. The pericardial cavity was opened after the removal of a thin layer of fatty thymus, and was found free of fluid and adhesions.

Heart, small sized and contracted ; weighs 190 gm.

Length of heart, 9 cm.

Muscle bundles of fair size and consistency and of greyish-brown colour. Sub-epicardial fat decreased in amount. All valves intact and normal in size and thickness.

Lungs : Right weighs 600 gm., voluminous, covered by smooth

glistening moist pleura of dark purple-greyish colour. It is interrupted here and there by accumulation of exogenous pigment. On cut section presents a dark purple surface throughout the three lobes of the lung, markedly increasing in consistency, and with numerous dark brownish patches scattered throughout the surface. On pressure, a frothy pinkish fluid is expressed from the alveoli bronchi. No scars, no calcareous deposits are made out.

Left lung: Weighs 500 gm.; presents a similar picture throughout its lower lobe, which is also markedly congested, showing several small patches of consolidation and permits the escape of a pinkish, sero-sanguineous fluid from the alveoli. The upper lobe of left lung is well aerated, of a bright pink colour, collapsed, showing no pathological changes whatsoever.

Liver: Weighs 1,100 gm.; below average in size; surface covered by thin, glistening capsule of a greyish-yellow colour. Cut surface is of a greyish-cream colour. Consistency markedly reduced, lobulation indistinct. Iodine test negative.

Spleen: Weighs 100 gm., small size capsule, thickened and wrinkled. On cut section surface is of a brownish-red colour. Trabecular and Malpighian corpuscles are distinct.

Kidneys: right weighs 120 gm.; left weighs 110 gm.

Both present similar picture. They are of small size. Capsule strips with ease, leaving a smooth greyish-yellow surface. Consistency markedly diminished. On cut section medulla is of normal size, cortex reduced in amount. Aside from a pale yellow colour, no definite pathological changes.

Adrenals: right rather well preserved; left shows a soft medulla with a congested central vein.

Gastro-intestinal tract: the stomach is normal.

Small intestines are collapsed; mucosa shows several patches of marked congestion, otherwise normal.

Large intestine is filled with greenish fluid, on the removal of which a thickened mucosa with numerous small, deep, clean ulcers scattered throughout the length of the large intestine. The ulcers measure from 3 to 5 mm. in diameter, irregular in outline, rather deep seated, base clean and glistening. They become more numerous in the lower portion of the large intestine.

Pancreas is normal.

Blood-vessels are rather strikingly free of arteriosclerotic changes, very elastic but somewhat reduced in circumference.

Bladder is markedly distended, muscular walls stretched, mucous membrane pale, showing a few petechial hæmorrhages.

Testes are small and soft; tubules easily pulled out of supporting tissue.

Prostate is apparently normal.

Neck organs: Right thyroid is twice the size of left, consistency normal. Three parathyroids measuring 5 mm. in diameter, oval in outline, were removed.

Larynx, normal.

Œsophagus, normal.

Brain is of moderate size, shows no apparent reduction in the prominence of the gyri. Several white opacities are seen in the meninges of the cerebral hemispheres. The vessels of the cortex and circle of Willis are normal.

Spinal cord was found to be of average size. Meninges appeared normal; there was no section evidence of degenerative atrophy in the posterior columns, but no other changes could be made out.

Microscopical examination of the viscera (Dr. J. H. Globus) :—

Lungs: Alveoli are crowded with red blood cells and polymorphonuclear leucocytes. The lining epithelium is desquamated, the blood-vessels in the walls are markedly congested. Bronchial tubes have lost their membrane lining, the lumen filled with desquamated epithelium—. polymorphonuclear leucocytes. The wall shows marked congestion of blood-vessels.

The liver is normal.

Spleen: trabeculæ prominent, walls of blood-vessels markedly thickened, Malpighian corpuscles in large numbers, fine size, blood-vessels engorged.

Kidneys, normal.

Pancreas, normal.

Adrenals, normal.

Thyroid: alveoli filled with pink staining colloidal matter. Lining epithelium of alveoli extremely thin. Their cellular structure can hardly be made out.

Spermatic cord, normal.

Prostate: alveoli are few in number, few concretions present.

Colon: several small abscesses opening on the surface of the mucosa are found extending deep into the submucosa.

Skeletal muscles: muscle fibres small in calibre, fragmented striations indistinct, nuclei few in number and pyknotic.

Anatomical Diagnosis.

Hypostatic congestion of lungs, lobular pneumonia, pulmonary œdema, fibrosis of spleen and ulcerative colitis.

Histological Examination of the Central Nervous System.

Blocks of tissue were taken from the cerebral cortex, the cerebellar cortex, the dentate nucleus, and the spinal cord for alcohol fixation and the Nissl method.

The brain and cord were fixed in formalin and various levels removed for cellular stains (toluidin blue, hæmatoxylin eosin). The remainder was then placed in Müller's fluid in preparation for the Weigert-Pal method.

Microscopic sections were prepared in the laboratory of the Montefiore Home and Hospital. The larger Weigert-Pal sections were made in the neurological laboratory of Columbia University, and I am much indebted to Professor Tilney for extending to me the courtesy of the laboratory in the preparation of this work.

Spinal cord: The large multipolar cells of the anterior horns are normal, at the cervical, dorsal and lumbo-sacral levels. The anterior roots are normal. The cells of the columns of Clarke in the dorsal region show signs of atrophy and are diminished in number.

The posterior columns show advanced atrophic change which may be traced from the lower lumbo-sacral levels to the nuclei of Goll and Burdach in the lower portion of the medulla (fig. 1).

The columns of Goll are almost completely atrophic, while those of Burdach show a lesser degree of involvement. The posterior cornua appears somewhat reduced in size. The fibres of Lissauer's tract appear fairly well preserved.

There are marked evidences of atrophy in the lateral columns, corresponding to the ventral and dorsal-cerebellar tracts (direct cerebellar and Gowers' tract). There is a lesser degree of diffuse atrophic change in that portion of the lateral columns corresponding to the crossed pyramidal tracts and the spino-thalamic tracts. In the area occupied by the crossed pyramidal tracts this is not clearly defined and as the Babinski reflex was absent it would seem likely, as suggested by Marie, that these atrophic fibres belong to other non-pyramidal systems. This would seem all the more probable as these slighter evidences of atrophy are limited to the cervical and dorsal regions and are scarcely at all demonstrable at the lumbar levels.

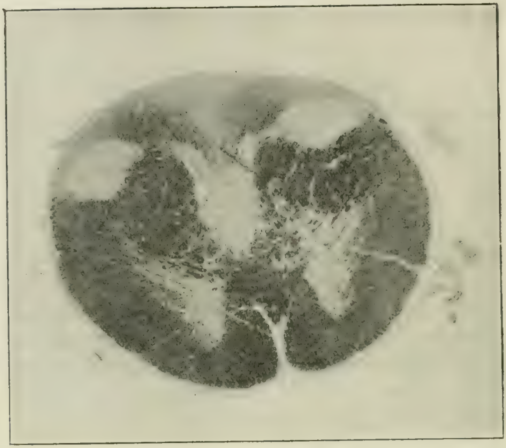


Fig. 1.—Cross section of the medulla oblongata, at the level of the pyramidal decussation. Weigert-Pal stain, showing atrophic changes in the tracts of Goll, Burdach and the spino-cerebellar tracts.

Fig. 2.—Medulla at the level of the inferior olive, showing atrophy of the spino-cerebellar tracts. The pyramidal tracts, the fillet, and the inferior olive and olivo-cerebellar fibres are well preserved.

In the anterior columns there is some marginal pallor, especially in the region of Helweg's tract. The direct pyramidal tracts are not atrophic.

The chief evidences of system involvement are therefore confined to the columns of Goll, Burdach, and the ventral and dorsal spino-cerebellar tracts. In both the cervical and dorsal regions there are, however, slight but rather diffuse atrophic changes.

There are no vascular lesions other than slight secondary thickening in the sclerosed areas, and no evidences of inflammatory lesions. The meninges are slightly thickened in the region of the sclerosed tracts but show no acute changes. The posterior roots show evidences of atrophy. The anterior roots are well preserved.

There is a combined degenerative atrophy of the postero-lateral columns of the cord. Fairly complete of the column of Goll, partial of the column of Burdach; and also of the direct cerebellar tract, Gowers' tract and of the columns of Clarke (cells and fibres). In addition to this there were slight diffuse atrophic changes in the lateral columns. The pyramidal tracts were not atrophic, which is supported by the clinical findings (absence of the Babinski reflex).

Medulla oblongata (fig. 2).—The cranial nerve nuclei of this region (12, 10, 9 and 8) are normal. The pyramids are of normal size and configuration, and there is no evidence of pallor in the Weigert-Pal specimens. The fillet is also normal in size and appearance. The direct cerebellar tracts show atrophic changes as they pass into the corpora restiforme.

The nuclei of Goll and Burdach as well as the fibræ arcuatæ internæ are well preserved.

The inferior olives and accessory olives are normal in size, general appearance and cellular content. The olivo-cerebellar fibres are well preserved.

The descending roots of the trigeminus and the solitary fasciculus are not atrophic.

The fasciculus longitudinalis posticus is normal.

The only noteworthy pathological alterations in this region are the evidences of atrophy posteriorly in the lower levels of the medulla where the tracts of Goll and Burdach pass into their respective nuclei and laterally correspond to the direct cerebellar tracts.

The large sensory and motor projection systems and the inferior olivary complex and its fibres of communication with the cerebellum are normal.

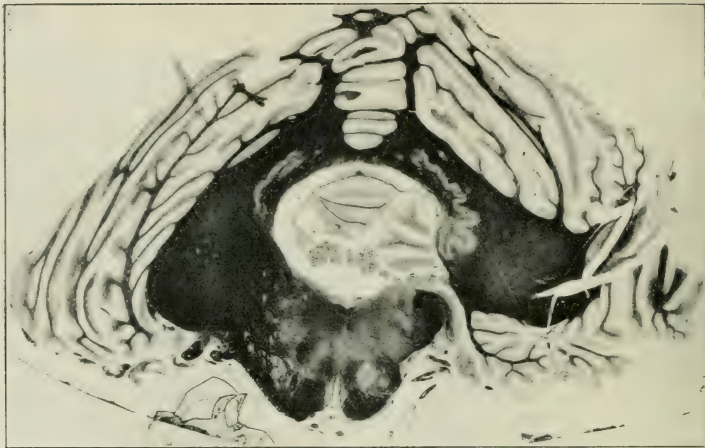


Fig. 3.—Vertical section through cerebellum and pons, at the level of the intrinsic cerebellar nuclei. The peduncles and arbor vitæ of the cerebellum are well preserved.

Fig. 4.—Cross section through the pons at the level of the trochlear decussation. Note the marked reduction in the fibres of the superior cerebellar peduncles and especially the atrophic changes in the region of the peduncular decussation. The pontine fibres constituting the stratum superficiale and the stratum profundum are well preserved.

Pons varoli (figs. 3 and 4).—The pontine nuclei, both ventral and dorsal groups, are normal and show no reduction in size or number of the cells. The fibres of the stratum superficiale pontis and the stratum profundum pontis and their continuation in the middle cerebellar peduncles are free from atrophic changes.

The corpora trapezoidii and the superior olives are normal. In higher levels of this region the superior cerebellar peduncles show distinct evidences of atrophy and considerable reduction in size. There are also atrophy and evidences of a marked diminution in the number of fibres at the decussation of the superior cerebellar peduncles.

The cranial nerve nuclei of this region are normal (eighth, seventh, sixth and fifth nerves).

The lateral fillet is normal and the fasciculus longitudinalis posticus is well developed on both sides. The essential pathological change in the pons is an atrophy of the superior cerebellar peduncles. The other structures of the pons, including its peduncles and the pontine nuclei, are normal in size and appearance.

Mid-brain (figs. 5 and 6).—This region reveals the atrophy of the fibres of the superior cerebellar peduncles by the diminished decussation of the peduncles which is so characteristic a feature of these levels. The lateral lemniscus and both anterior and posterior colliculi are normal in appearance.

In the higher levels the ventral decussation of Forel appears apparently in full strength ; so while there is a distinct and well-marked atrophy of the fibres passing between the dentate nucleus and the red nucleus this is apparently not continued over into the rubro-spinal tract, which receives further confirmation by the preservation of the cells of the red nucleus.

The various oculo-motor nuclei and root fibres are normal. The cells of the locus niger and of the nucleus ruber appear normal and so far as could be determined there was no reduction in their size or number. Especially interesting is the normal condition of the long projection tracts of the crus cerebri, viz., the fronto-ponto-cerebellar, the pyramidal and parieto-temporo-occipito-cerebellar tracts. The ponto-cerebellar tracts show no evidences whatever of atrophy or pallor.

The only noteworthy change in the mid-brain was the well-marked atrophy of the fibres which enter into the decussation of the superior cerebellar peduncles. I would also emphasize the normal appearance of the connections between the cerebrum and the cerebellum, viz., the fronto-ponto and the parieto-temporo-occipito-ponto cerebellar tracts.

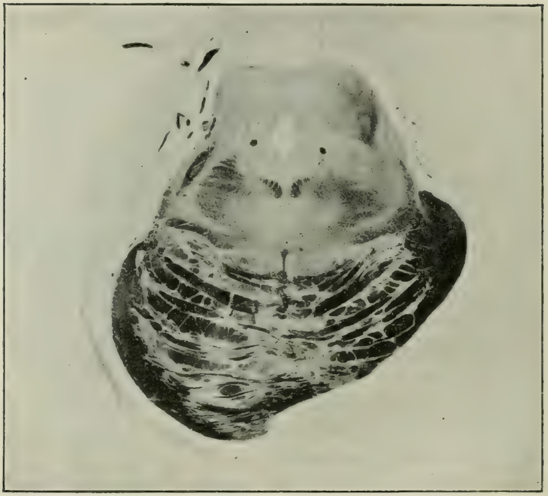


FIG. 5.—Cross section through the inferior corpora quadrigemina, at the level of transition from pons to midbrain. Note the marked reduction in size and the atrophic changes in the region of the superior cerebellar peduncles and their decussation. The stratum superficiale and stratum profundum of the pons are well preserved.

FIG. 6.—Cross section through the superior corpora quadrigemina. The long projection fibres of the crura cerebri are well preserved, including the fronto-cerebellar and the parieto-temporo-occipito-cerebellar tracts. Note diminution of peduncular fibres in the region of the red nucleus.

Fig. 7.—Cross section through the interbrain showing the corpus striatum and optic thalamus.

Fig. 8.—Section through the cerebellar hemisphere showing normal arbor vitæ and cerebellar cortex.

Interbrain (fig. 7).—Sections of the interbrain reveal no abnormalities of the optic thalamus or of the corpus striatum. The cells of the locus niger and nucleus ruber are well preserved and are normal in number and appearance as are those of the corpus subthalamicum. There is no apparent atrophy of the fibre systems of either the optic thalamus or corpus striatum.

Cerebral cortex.—Sections of the cerebral cortex from the frontal, parietal, temporal and occipital areas were examined by cellular and fibre stains and revealed no noteworthy abnormality.

The meninges and blood-vessels showed no special pathological changes.

Cerebellum (fig. 8).—The general histological appearance and markings of the cerebellum are quite normal. The arbor vitæ is well developed and the grey matter of the cortex is normal. The molecular, Purkinje, and granular layers of the cerebellar cortex present a normal histological appearance. The large cells of Purkinje are normal in size and number.

Vertical sections were made through the cerebellum including the structures of the pons and medulla oblongata. In these sections were included the intrinsic nuclei of the cerebellum.

The convolutions of the nucleus dentatus appear a little less voluminous than is normal and the cerebellar olive gives the impression of being somewhat reduced in size. The nucleus emboliformis, globosus and tecti are present and of normal size and appearance.

Histological study shows a considerable atrophy and diminution in number of the cells of the dentate nucleus (figs. 9, 10, 11, 12). Many of these cells appear shrunken, rounded and atrophic, and a large number have completely disappeared. An attempt was made to estimate in some degree the cellular loss by making comparative cell counts with normal specimens. By this method the loss in the number of cells varied from one-half to one-third.

Because of the small size of the other intrinsic nuclei of the cerebellum (nucleus tecti, emboliformis and globosus) it was not possible to make the same accurate estimation of the cell content as in the case of the larger nucleus dentatus.

A study of the sections, however, revealed little, if any, evidence of cellular loss or atrophy. The cells of the nucleus tecti especially were numerous and well preserved. The chief cellular changes were confined to the convolutions of the cerebellar olive (corpus dentatum). The meninges and vascular structures of the cerebellum were normal. Only

within the nucleus dentatum did the blood-vessels show some secondary thickening.

The cerebellum was, therefore, normal in its histological appearance, with the sole exception of an extensive and well-marked atrophy of the cells of the dentate nucleus.

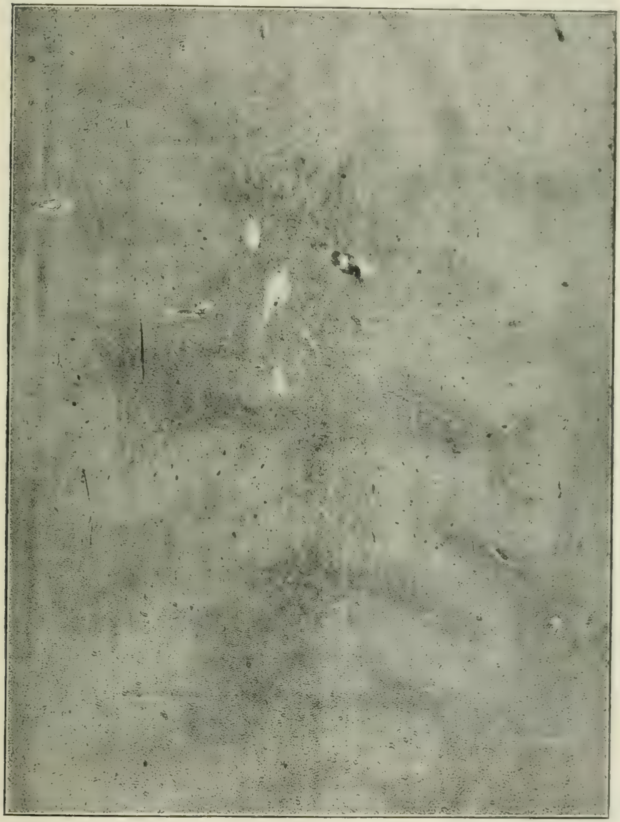


FIG. 9.—Dyssynergia cerebellaris myoclonica. Primary atrophy of the dentate system. Hæmatoxylin-eosin stain, section of the corpus dentatum, showing the convolution of the cerebellar olive and the great reduction in size and number of the cells of the corpus dentatum.

Summary of Pathological Changes.

The histological study of the central nervous system in this case showed the typical spinal cord changes of Friedreich's ataxia. There was atrophy of the posterior columns of the cord which could be traced

FIG. 10.—Normal control: same magnification and stain as fig. 9. Section through the corpus dentatum, showing convolution of the cerebellar olive and normal appearance of the cells of the corpus dentatum.

to the nuclei of Goll and Burdach in the medulla. Atrophic chang were also present in the direct cerebellar tract of Flechsig and t tract of Gowers (ventral and dorsal spino-cerebellar tracts). T

columns of Clarke were atrophic, and there were also some changes in the area occupied by the spino-thalamic tracts.

There was no definite atrophy of either the direct or crossed pyramidal tracts and the slight pallor in certain areas I would ascribe to

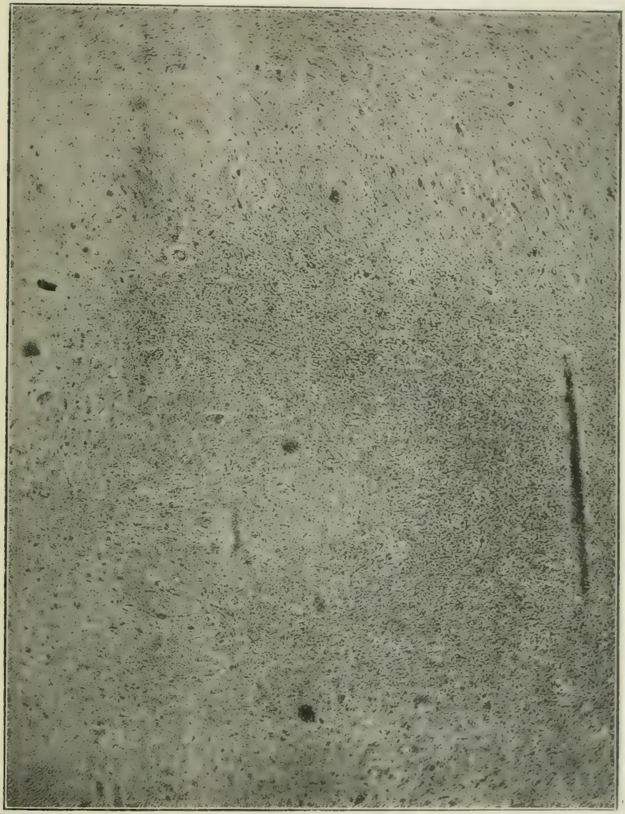


FIG. 11.—Hæmatoxylin-eosin. The corpus dentatum of dyssynergia cerebellaris myoclonica showing marked atrophy and diminution in number of the cells of the cerebellar olive. (Primary atrophy of the dentate system.)

degeneration of other non-pyramidal fibres which belong to this region.

Histological study of the brain-stem and cerebellum showed the

following conditions: There was an extension into the medulla of the system atrophies of the cord, viz., the direct spino-cerebellar tracts. The atrophy of the posterior columns apparently ceased at their ter-

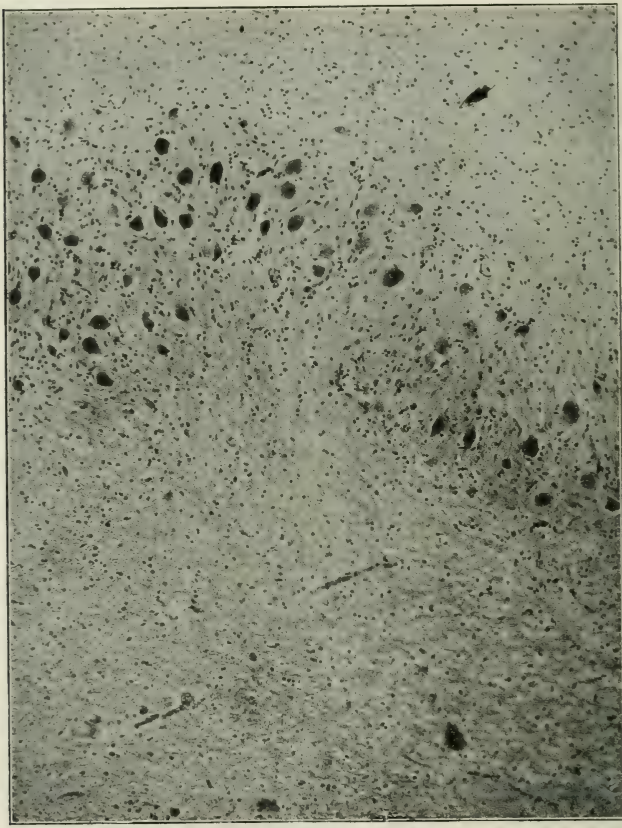


FIG. 12.—Normal control. Same magnification and stain as fig. 11. Showing normal appearance of cells of the corpus dentatum.

mination in the nuclei of Goll and Burdach, for the fibræ arcuatæ internæ were well preserved and the corpus restiforme well developed.

The other important and essential lesion was an atrophy of the

motor cells of the corpus dentatum of the cerebellum and of their efferent neurones in the superior cerebellar peduncles. There was no atrophy of the other cerebellar systems, and none of the nucleus ruber. The lesion was therefore confined to this short and very important internuncial common path which conveys the motor impulses of cerebellar origin to the spinal pathways.

The lesions were therefore both spinal and cerebellar. The spinal lesion is that common to Friedreich's ataxia; the cerebellar lesion I would regard as a special form of system disease, viz., primary atrophy of the efferent dentate system of the cerebellum.

Chapter IV.—Primary Atrophy of the Efferent Dentate System and its Relation to Dyssynergia Cerebellaris Progressiva (Progressive Cerebellar Tremor).

Anatomical Considerations.

The cerebellum is an extremely complex ganglionic structure, having massive and important connections with the spinal cord, brain stem and the cerebrum. It has an elaborate convoluted cortical structure, composed of various cellular and association layers, as well as commissural systems and peduncles. The inferior and middle peduncles are composed of afferent fibres on their way to the cerebellar cortex.

The inferior cerebellar peduncle or corpus restiforme consists of two divisions, a spinal and a bulbar. Among its constituents are the direct cerebellar tract; the anterior and posterior arcuate fibres of the nuclei of Goll and Burdach; olivo-cerebellar fibres; fibres from the nucleus lateralis and nucleus arciformis of the medulla; and the nucleo-cerebellar tracts consisting of fibres passing from the cranial nerve nuclei of the pons and medulla.

The chief constituents of the middle cerebellar peduncle or brachium pontis are: (1) the fronto-ponto-cerebellar tract; (2) the temporo-ponto-cerebellar tract; (3) the occipito-ponto-cerebellar tract; and (4) the parieto-ponto-cerebellar tract. According to Tilney and Riley [6] these fibres originate in cells of the frontal, temporal, occipital and parietal lobes respectively. They descend through the internal capsule and the cerebral peduncle to end in the cells of the pontine nuclei. From these cells arise the ponto-cerebellar fibres which decussate in the pons and pass to the cerebellar hemisphere of the opposite side.

The superior peduncle, on the other hand, is essentially efferent and

motor in function, providing the principal pathway for the distribution of cerebellar impulses to the muscular system.

This system of fibres, together with its fellow of the opposite side, undergoes a complete decussation before reaching the red nucleus of the midbrain. Its chief constituents are the cerebello-rubral tract and the cerebello-thalamic tract. It also contains a cerebello-tegmental tract.

The efferent fibres composing the superior cerebellar peduncle arise in cells of the intrinsic nuclei of the cerebellum. These are the dentate nucleus, the nucleus globosus, the embolus, and the nucleus tecti. Efferent fibres from the nucleus tecti also pass in the juxta-restiform body to the motor cranial nerve nuclei of the brain-stem (cerebello-nuclear fibres). The dentate nucleus is related to the neocerebellum or hemispheric system. Phylogenetically, it coincides in development with the cerebral cortex. The other intrinsic nuclears are older and are related to the paleocerebellum or vermian system.

All of the efferent fibres arising in cells of the intrinsic nuclei I would include under the general heading of the efferent dentate system. Those originating in the paleocerebellar nuclei may be distinguished as the *paleodentate system* and those belonging to the dentate nucleus proper as the *neodentate system.*

This would simplify our existing nomenclature for the various constituents of the efferent cerebellar system (cerebello-rubral, cerebello-thalamic, and cerebello-tegmental system), and is justified by their morphological and functional identity. The term *dentate* is chosen, as the various efferent fibre systems of the cerebellum all take their origin in the intrinsic nuclei of the cerebellum, of which the dentate is the chief constituent. This, the more recent acquisition in the phylogenetic sense, represents the neodentate system, in contradistinction to the paleodentate system which takes its origin in the older nuclei.

Pathological Considerations.

In Chapter II I have described in some detail a group of cases characterized by symptoms of a progressive cerebellar disorder, associated with myoclonus-epilepsy.

In Chapter III a similar group of cases was reported in association with Friedreich's ataxia. In one of these cases with symptoms of a cerebello-spinal involvement, a careful histological study was made of the central nervous system, with the following results:—

The spinal cord showed the typical lesions of Friedreich's ataxia, which represented the pathological basis for the spinal symptoms of the disorder. A careful examination of the cerebellum and its connections revealed in addition an atrophy of the cells of the dentate nucleus and of the superior peduncles of the cerebellum.

The nature of the atrophic process and the strict limitation of the lesion to a special group of neurones are characteristic of a system disease. This condition I believe is a primary atrophy of the efferent dentate system of the cerebellum, and the cause of the cerebellar portion of the symptomatology.

Atrophic changes in the cells of the dentate nucleus in cases of Friedreich's ataxia have been previously described by Spiller [20], and also by Thomas and Durupt [4], in association with other lesions of this region. In the case recorded by Thomas and Durupt there were in addition to atrophy of the dentate nucleus pathological changes in the red nucleus, the fillet and the posterior corpora quadrigemina.

Like the spinal cord, the cerebellum shows a special tendency to various types of congenital defects and system diseases. These may be limited to the cerebellum but are often combined with spinal cord changes, and represent a group of cerebellum-spinal disorders. Prominent among these are the hereditary spinal ataxia of Friedreich, and the hereditary cerebellar ataxia of Marie, which may be present alone or in combination. In this disorder the lesion of the cerebellum is usually a general atrophy of the whole organ, which appears much reduced in size.

In addition to this more general type of cerebellar atrophy many more circumscribed forms are recognized, in which the lesions are limited to certain special systems of the cerebellar mechanism.

There is, for example, the olivo-ponto-cerebellar atrophy of Dejerine and Thomas [6] in which the atrophic lesions are confined to the inferior olive and its cerebellar connections, the pontine nuclei, middle peduncles of the cerebellum and the cerebellar cortex. All of the intrinsic nuclei of the cerebellum and the superior cerebellar peduncles are intact. This type is neither congenital, hereditary nor familial and appears only in later life.

Then there is a type described by Gordon Holmes [9], the olivo-cerebellar type of cerebellar atrophy. In this form there is atrophy of the cerebellar cortex, the inferior olive and the olivo-cerebellar tract. The pontine nuclei and middle peduncles, and the intrinsic nuclei and superior cerebellar peduncles are normal. This form differs from the preceding in its familial incidence.

Still another type has been added by Le Jonne and Lhermitte [15], the olivo-rubro-cerebellar atrophy. This form is characterized by atrophy of the inferior olive and olivo-cerebellar fibres, but also include the intrinsic nuclei, the superior cerebellar peduncles and the cells of the nucleus ruber. The structure of the cerebellar cortex, the pontine nuclei, and middle peduncles are intact.

The lesions in all of these types are those of simple atrophy which characterize a system-disease, and consist of primary degeneration of the parenchyma with secondary proliferation of glia.

In addition to the cerebellar system diseases there are also pure cortical types of cerebellar atrophy in which the degenerations are limited to the finer structures of cerebellar cortex (cells of Purkinje and granular layer). Such cases have been described by André-Thomas [1] and Rossi [17], and present the typical clinical picture of cerebellar disease.

At the present time we may recognize therefore the following pathological types of cerebellar atrophy. The cerebellar cortical type of André-Thomas, the olivo-ponto-cerebellar type of Dejerine and Thomas; the olivo-cerebellar type of Holmes, and the olivo-rubro-cerebellar type of Le Jonne and Lhermitte.

To these various types I would add the primary atrophy of the dentate system (figs. 9 and 11). This is a system disease characterized by atrophy of the cells of the dentate nucleus (neodentate system) and thinning of the superior cerebellar peduncles. All other important structures of the cerebellum, the cortex, the olivo-cerebellar and ponto-cerebellar systems are intact. In this group of cases the symptoms of a cerebellar disorder are much more evident in the co-ordinated movements of the extremities than in those of the trunk. The cerebellar or intention tremor which was present in greater or lesser degree in all of the cases was a characteristic symptom. The dyssynergia is, therefore, of the appendicular rather than the axial type. In this respect differing from the massive trunkal involvement of many other forms of cerebellar atrophy.

There already exists in medical literature a number of isolated facts tending to show the close connection between, what I term, the dentate system and the cerebellar or intention tremor. The relation of the intention tremor to the cerebello-rubral system was emphasized some years ago by Gordon Holmes [8], based on numerous clinical and pathological observations. Sander [18] and Touche [22] have also reported cases of intention tremor in association with lesions of the

dentate nucleus. In the experimental field, the work of Ferrier and Turner [7] on monkeys has also shown the relation of the cerebellar or intention tremor to the efferent cerebellar system. After section of the superior cerebellar peduncles these investigators observed: "Except when quite at rest, a constant tremor in the homolateral arm and leg which passed into larger oscillation on volitional effort. The limbs were the subjects of fine tremors which became amplified on exertion."

As the pathological changes in the case just recorded are limited to the neodentate system of the cerebellar mechanism, it is but natural that the more recently acquired and more highly differentiated motor activities should show the greatest degree of involvement. It is for this reason I believe that the affection is predominantly appendicular in its manifestations and the intention tremor so conspicuous a symptom, the neodentate portion of the efferent cerebellar system showing a greater vulnerability to abiotrophic degeneration.

In this respect, there is a certain similarity to another system disease which I have described in relation to the corpus striatum, viz., the primary atrophy of the efferent pallidal system [11].

This disease represents a *primary* or *essential* form of paralysis agitans. The pallidal system, like the dentate system, is a common pathway and serves for the transmission of all motor impulses from the corpus striatum. In this form of paralysis agitans the earliest involvement is usually of the extremities, which is probably due to the initial atrophy of the pallidal cells of the neostriatum (neopallidal cells). I believe, therefore, in the cerebellum as well as in the corpus striatum we must recognize the existence of an internuncial common pathway for the transmission of efferent impulses. Both systems may be the seat of abiotrophy in the sense in which this term was used by Gowers. One is the primary atrophy of the pallidal system causing the primary or essential form of paralysis agitans. The other is primary atrophy of the dentate system causing the primary or essential form of dyssynergia cerebellaris progressiva. In addition to these manifestations of primary atrophy, secondary and symptomatic forms may be recognized, caused by tumours, inflammatory and vascular lesions.

Chapter V.—The Relation of the Cerebellum to the Static or Posture System of Motility.

All investigators have recognized the important rôle played by the cerebellum in the control and regulation of motility, its influence on muscle tonus, equilibrium and the co-ordination of movement. The trend of modern opinion, under the leadership of Babinski, has been to group all cerebellar symptoms under the general heading of synergia. According to this conception, which is now generally accepted, the essential function of this important structure is the regulation of the synergies of movement, and synergia is regarded as the principal function of the cerebellum. And yet, notwithstanding this recognized rôle in the control of movement, there is much that the prevailing theories of cerebellar function have failed to explain.

In the course of previous investigations on the subject of motility I reached the conclusion that the efferent nervous system is susceptible of division into two separate components which I termed *the static and kinetic systems* [12] [13]. As this conception has an important bearing on the interpretation of cerebellar symptoms a brief outline of its chief features may be given here.

According to this view, motility is subserved by two distinct components, each represented throughout the entire efferent nervous system by separate neural mechanisms, which are physiologically and anatomically distinct. One is the movement proper, which is subserved by the *kinetic system* (motion system). The other represents that more passive form of contractility which we recognize in tonus, posture and equilibrium and is subserved by the *static system* (posture-system).

The term static is used to designate that peculiar property of the muscle fibre by which it becomes fixed in posture. In the reflex nervous system this is manifested by postural tonus and at the higher levels by various postures and attitudes.

There are many reasons for the assumption that the kinetic system is related to the anisotropic discs and the static system to the sarcoplasm of the muscle fibre, as these elements differ in structure, innervation, mode of contractility and metabolism.

There are also reasons for the assumption that the transformation of movement into posture is effected by fixation of the sarcoplasm which converts the contractile muscle fibre from a kinetic into a static mechanism.

Therefore, movement is subserved by a kinetic, aud posture by a

static mechanism, the two systems supplementing one another and working together in harmony. For every movement starts from posture, is accompanied by posture, and terminates in posture, posture following movement like a shadow. At the same time, the postural mechanism exercises a stabilizing influence upon the course of movement itself.

Motility as it is observed in animal life may be divided more or less arbitrarily into three distinct groups, viz., reflex, automatic-associated, and isolated-synergic or dissociated types of movements.

All of these forms of movement are intimately associated with that other component of motility which we term posture, and the same postural groups may be recognized as in the classification of movement, viz., *reflex postures*, *automatic-associated postures* and *isolated-synergic types of posture* corresponding to their respective motion mechanisms. And it may be stated as a general principle that there is no form of motility from the simple reflex to the most skilled and individualistic type of movement which functions without a corresponding static or posture mechanism.

The essential integrating and correlating mechanism for the control of the static function of motility I believe to be the cerebellum. Afferent impulses from the periphery and efferent impulses from the cerebral cortex pass to this organ before their final distribution by way of the cerebello-spinal system to the posturing or static mechanism of skeletal muscles.

The older cerebellum stands in relation to what may be termed the *paleostatic system* which controls the older static or postural functions of "automatic and associated type." It takes its origin in the older nuclei of the vermis cerebelli (N. fastigii, globosus, emboliformis).

The cerebellar hemispheres, on the other hand, regulate the higher postural functions of motility through the medium of a neostatic system, which takes its origin in the cells of the dentate nucleus. The neostatic system is controlled from the cerebral cortex by special tracts which connect the various regions of the cerebral cortex (frontal, parietal, temporal and occipital) with the dorsal and ventral nuclei of the pons varolii, and thence to the opposite hemisphere of the cerebellum.

In posture, as in movement, no sharp line of demarcation can be drawn between the various types, and one group merges imperceptibly into another.

It is interesting to observe that all posture systems, *neostatic* as well as *paleostatic*, pass to the cerebellum for final integration and

co-ordination. This is in accord with the nature of the posturing mechanism and its secondary and unconscious rôle in motility. For while the higher forms of movement are initiated as conscious and voluntary processes, the corresponding postures are secondary, and follow automatically in the path of movement.

The cerebellum, then, is an organ which is engaged in the control and regulation of the static or *posture synergies* of motility. In it are represented the infracortical centres for the posturing mechanism which are under both spinal and cerebral control. I would, therefore, regard the cerebellum as a central ganglionic station for the co-ordination and control of static or posture synergies—in contradistinction to kinetic or motion synergies which are localized in their respective kinetic spheres (rolandic and striatal). The static synergies are manifestations of the sarcoplasm of the muscle fibre and the kinetic synergies of the anisotropic discs. The static or posture mechanism according to this conception is concerned in the regulation of muscle tonus, the maintenance of postures and attitudes, and is also an important factor in guiding, checking and fixation of movement.

A striking experiment cited by André-Thomas [2], may be mentioned in confirmation of this point of view. A dog whose cerebellum had been removed was unable to stand or walk. When, however, he was thrown into the water he could swim without difficulty. This peculiar phenomenon I would explain as follows: When the animal attempted to stand or walk, the posturing mechanism was essential to co-ordinated movement, to counteract the force of gravity. In a supporting fluid medium like water, however, posture synergies are of secondary importance, and as the kinetic system with its synergies are undisturbed the act of swimming takes place without difficulty.

In many respects, this theory of posture synergy also harmonizes with our modern conception of cerebellar symptomatology. In the co-ordination of all forms of movement, posture synergy is of prime importance. Loss of this control is, I believe, the cause of the scanning speech, cerebellar ataxia, intention tremor, hypermetria, adiadokokinesis and nystagmus which we observe in cerebellar disease.

In cerebellar ataxia, for example, a characteristic feature is the decomposition of movement which takes place during the execution of a co-ordinated act. When this is present it requires several disjointed movements to accomplish what is normally performed as a continuous one. The discontinuity of movement, which is one of the cardinal

symptoms of cerebellar disease, may be referred to a disorder of posture synergy which prevents the posture and motion systems from acting together in harmony. The decomposition of movement represents therefore an effort to compensate for the loss of the posturing mechanism.

Hypermetria and dysmetria of cerebellar origin may also be explained by a failure of the posture system in its function of guiding and checking movement. For in any co-ordinated act, posture formulæ are as necessary as motion formulæ and play an important rôle in giving stability and direction to movement. The checking of movement and its fixation in posture is also an essential function of the static system.

Adiadokokinesis may likewise be ascribed to a disorder of the static mechanism, the rapid succession movements which characterize this disorder being dependent upon quick fixation and sudden release of the posture mechanism.

The cerebellar or intention tremor is a particularly striking example of a disorder of the posture mechanism. When a patient presenting this symptom attempts a movement, the extremity passes into coarse ataxic oscillations which increase in intensity as the object is reached and the extremity tends to become fixed in posture. There is a coarse ataxia and tremor-like oscillation not only during the passage of the movement, but also during attempts at fixation. It is particularly at the end of the movement when the extremity tends to become fixed in posture that the intention tremor is often most active. This characteristic of the intention tremor is due to a posture asynergia, and serves to differentiate it from other forms of purely kinetic ataxia.

Cerebellar nystagmus may also be mentioned as representing a similar mechanism. Here again the movements increase when the attempt is made to fix them in posture.

Very interesting is the influence of the vestibular mechanism on the posture system of skeletal muscles. Barany has shown that all movements, trunkal and appendicular, are under a certain labyrinthine control. This is shown by the nystagmus, the pass-pointing and falling tests.

In brief then, while asynergia may be regarded as the fundamental symptom of cerebellar disease, I would limit this conception to *posture asynergia*, which is a disorder of the static or posture system of motility.

Posture asynergia may be paleostatic or neostatic, depending upon the portion of the cerebellum involved. Paleostatic asynergia is a disorder of lower forms of posture of automatic-associated type, and is largely trunkal or axial in distribution. Neostatic asynergia, on the other hand, is a disorder of the higher forms of posture, of isolated synergic type and is essentially appendicular in distribution. One is a spinal, the other a cerebral form of asynergia.

In the system disease of the cerebellum described in the previous chapter—"Primary Atrophy of the Dentate System"—the atrophic changes were limited to the cells of the corpus dentatum (neodentate system). This is a system which is related to the neocerebellum and its recently acquired functions. For this reason the cerebellar symptoms of this disorder are predominantly appendicular in distribution, the extremities representing in the realm of posture, as in movement, the highest degree of development.

Chapter VI.—Concluding Remarks.

In the previous chapters, under the title, "Dyssynergia Cerebellaris Myoclonica," I have described what appears to be a well-defined type of nervous disease, presenting the clinical picture of a progressive cerebellar disorder in association with myoclonus-epilepsy. Four of the cases were sporadic and no history could be elicited of any hereditary or familial tendency to either cerebellar disease or myoclonus-epilepsy.

The cerebellar disturbance affected more especially the finer movements of the extremities and the dyssynergia was predominantly appendicular in distribution. The cerebellar or intention tremor was a marked feature of all the more advanced cases, in this respect resembling the clinical group which I described some years ago under the title "Dyssynergia Cerebellaris Progressiva," or "Progressive Cerebellar Tremor."

The myoclonus-epilepsy, in its general manifestations, was very similar to the disorder as described by Unverricht [23]. The familial incidence, however, which is one of the chief characteristics of the Unverricht type, was lacking, excepting in the group associated with Friedreich's ataxia. The myoclonus-epilepsy, therefore, resembles in some respects the type of the disorder described by Rabot [16]. This is a sporadic form, beginning somewhat later in life than the Unverricht type and in which the myoclonus is intermittent in character and less

progressive in its evolution. Fundamentally, however, both types are very similar and are united by transition forms.

The relation of the cerebellar disorder to myoclonus-epilepsy in the group of cases which I have described is quite obscure and in the present state of our knowledge but little light can be thrown on this question. It is quite possible that the combination is only accidental and represents the association of two independent nervous disorders in a predisposed individual. Such combinations in the realm of neuropathology are by no means uncommon. Nevertheless, I think that one should not be too hasty in concluding that here is a mere combination of separate clinical entities. We know so little of the underlying cause and pathology of myoclonus and its relation to the various structures of the central nervous system that the possibility of a form related to the static or posture system should be considered. It is conceivable, for example, that sudden breaks in the continuity of postural control or synergy might express themselves in terms of compensatory movements of a myoclonic type.

Interesting in this connection are the peculiar epileptiform attacks which occurred in two of the cases, which I have termed static seizures. These attacks are characterized by sudden loss of postural control without convulsive manifestation and are accompanied by slight transitory obscuration of consciousness. The patient without warning plunges suddenly to the ground, apparently from a sudden cessation of postural control. These attacks are so sudden and the fall so immediate that injury is very likely to ensue. There are no muscular spasms and the patient rises almost immediately. The blurring of consciousness is often only momentary; indeed in some of the attacks consciousness is apparently unaffected.

In addition to the clinical type, termed dyssynergia cerebellaris myoclonica, a similar complex of symptoms was observed in another group of cases associated with the spinal symptoms of Friedreich's ataxia. These, however, were of the family type, two cases occurring in one family. Mention was also made of an observation recorded by Boschi showing that such combinations of symptoms, while rare, are not unknown in medical literature.

In one case, belonging to this group, a careful histological study was made of the central nervous system, which revealed the typical spinal lesions of Friedreich's ataxia, in conjunction with a peculiar type of system atrophy of the cerebellum, which I termed primary atrophy of the dentate system. The atrophic changes involved only the cells of the

corpus dentatum and the fibres of the superior cerebellar peduncles. The pathological changes were therefore limited to the cells and fibres of a special system of neurones, which because of their relation to the dentate nucleus was termed the dentate system. All other structures of the cerebellum were normal.

To the other recognized system atrophies of the cerebellum, viz. : the cerebellar cortical atrophy, the olivo-ponto-cerebellar atrophy; the olivo-cerebellar atrophy and the olivo-rubro-cerebellar atrophy, I would add the atrophy of the dentate system, which is a primary atrophy of its chief efferent system. The association of this form of cerebellar atrophy with Friedreich's ataxia is not surprising in view of the frequency of cerebello-spinal combinations.

The cerebellar portion of the symptomatology of dyssynergia cerebellaris myoclonica I would refer to the atrophy of the efferent dentate system. In the present state of our knowledge it is difficult, if not impossible, to refer general cerebellar symptoms to special parts of the cerebellar mechanism. And it is generally held that the various types of cerebellar atrophies mentioned above cannot be distinguished with certainty on the basis of their cerebellar symptomatology alone. In other words, asynergia is the characteristic symptom of cerebellar disease, no matter which portion of the cerebellar mechanism is involved.

It is, therefore, with some hesitation that I suggest the early appearance and predominance of appendicular asynergia and especially the cerebellar tremor as perhaps characteristic of this type of cerebellar disease (primary atrophy of the dentate system) and yet these were the characteristic features of the clinical type under consideration.

Some years ago I described a primary system disease of the corpus striatum which was associated with the symptomatology of paralysis agitans. The lesions consisted of atrophy of the large motor or pallidal cells of the corpus striatum, and of their efferent fibres in the ansa system. The disorder was called primary atrophy of the pallidal system. This I regarded as the primary and essential form of paralysis agitans and all other clinical manifestations of the disease as secondary or symptomatic in nature due to vascular, inflammatory and neoplastic lesions.

A similar differentiation may be made in the case of the cerebellum and its efferent pathway, the dentate system. This may be the seat of a primary atrophic process producing the characteristics of a cerebellar disorder (primary atrophy of the dentate system). This system may

also be the seat of secondary or symptomatic involvement from tumours, inflammations and vascular lesions. The intention tremor of multiple sclerosis is a symptomatic manifestation of involvement of this system.

Primary atrophy of the pallidal system is, therefore, a disorder of a kinetic mechanism, giving rise to the symptomatology of paralysis agitans. Primary atrophy of the dentate system is a disorder of a static mechanism, giving rise to the symptomatology of dyssynergia cerebellaris.

In the final chapter I presented my views on the functions of the cerebellum and its relation to the static system of motility. According to this conception the function of the cerebellum is the control of the posture synergies of movement. Motility is subserved by two components throughout the whole of the efferent system, a kinetic or motion system and a static or posture system, both working together in harmony.

Motility, operating through its contractile mechanism, is therefore a compound of movements and postures. Movement is the active component which propels the organism or parts of the organism in space, the peripheral contractile mechanism of which is represented in the anisotropic discs of the striated muscle fibre. Posture is that more passive component which is represented in tonus and attitude. It tends to counteract the forces of gravity and the effects of atmospheric pressure and maintains the organism in a state of static equilibrium. The peripheral organ of this mechanism is the sarcoplasm of the muscle fibre. This system also plays an important rôle in the guiding, checking and fixation of movement. The various symptoms of cerebellar disease which are grouped under the general heading of asynergia may be attributed to a disorder of this mechanism.

BIBLIOGRAPHY.

[1] André-Thomas. "Atrophie lamellaire des cellules de Purkinje," *Revue Neurol.*, 1905, **13**, 917.

[2] *Idem.* "Cerebellar Functions," *Journ. of Nerv. and Ment. Dis.*, Monograph Series, New York, 1912, Chap. 11.

[3] *Idem.* "Le cervelet" (Case 5), Paris, 1897.

[4] André-Thomas and Durupt. "Examen du névraxe dans un cas de maladie de Friedreich," *Revue Neurol.*, 1912, **24**, 317.

[5] Boschi, Gaetano. "Ataxie héréditaire avec paramyoclonus multiplex; type Unverricht," *Journ. de Neurol.*, 1913, **18**, 141.

[6] Dejerine and Thomas. "L'atrophie olivo-ponto-cérébelleuse," *Nouv. iconog. de la Salpêtrière*, 1900, **13**, 330.

[7] Ferrier and Turner. "A Record of Experiments illustrative of the Symptomatology and Degenerations following Lesions of the Cerebellum and its Peduncles in Monkeys," *Phil. Trans. Roy. Soc. of Lond.*, 1894, **184**, (B), 719.

[8] Holmes, Gordon. "Certain Tremors in Organic Cerebral Lesions," *Brain*, 1904, **27**, 327.

[9] *Idem.* "A Form of Familial Degeneration of the Cerebellum," *Brain*, 1907, **30**, 466.

[10] Hunt, Ramsay. "Dyssynergia Cerebellaris Progressiva: A Chronic Progressive Form of Cerebellar Tremor," *Brain*, 1914-15, **37**, 247.

[11] *Idem.* "Primary Atrophy of the Pallidal System: A Contribution to the Functions of the Corpus Striatum," *Brain*, 1917, **40**, 58, and *Arch. Int. Med.*, 1918, **22**, 647.

[12] *Idem.* "The Static and Kinetic Systems of Motility," *Arch. of Neur. and Psych.*, 1920, **4**, 353.

[13] *Idem.* "The Static or Posture System and its Relation to Postural Hypertonic State of the Skeletal Muscles," *Neurol. Bull.*, 1921, **3**, 207.

[14] Jendrassik. "Hereditäre Krankheiten," Lewandowsky's "Handbuch der Neurol.," **2**, 394.

[15] Lejonne and Lhermitte. "Atrophie olivo-rubro-cérébelleuse," *Nouv. iconog. de la Salpêtrière*, 1909, **22**, 603.

[16] Rabot. "De la myoclonie épileptique," *Thèse de Paris*, 1899.

[17] Rossi. "Atrophie primitive parenchymateuse du cervelet à localisation corticale," *Nouv. iconog. de la Salpêtrière*, 1907, **20**, 66.

[18] Sander. "Ein pathologisch-anatomischer Beitrag zur Function des Kleinhirns," *Deut. Zeit. f. Nervenheilk.*, 1898, **12**, 363.

[19] Sioli. "Ueber histologischem Befund bei familiärer Myoklonus-Epilepsie," *Arch. f. Psychiat.*, 1913, **51**, 30.

[20] Spiller. "Friedreich's Ataxia," *Journ. of Nerv. and Ment. Dis.*, 1910, **37**, 411.

[21] Tilney and Riley. "The Form and Functions of the Central Nervous System," 1921, 456.

[22] Touche. "Deux cas de ramollissement du cervelet," *Revue Neurol.*, 1900, **8**, 149.

[23] Unverricht. "Ueber familiäre Myoclonie," *Deut. Zeit. f. Nervenheilk.*, 1895, **7**, 33.

ON DISORDERS OF MOVEMENT RESULTING FROM LOSS OF POSTURAL TONE, WITH SPECIAL REFERENCE TO CEREBELLAR ATAXY.

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CHAPTER I.—INTRODUCTION.

THE problems presented by cerebellar lesions, whether experimentally produced or the result of disease in man, have been the subject of much earnest research ever since Flourens published his classic observations a century ago. Perhaps no part of the nervous system has been more intensively studied than the cerebellum, and yet the final analysis of its symptom-complex and the determination of its functions have both so far eluded us.

When we consider the complexity of the physiological problem involved, it is perhaps not surprising that we have not been able to determine the functions of this organ, but it is more noteworthy that a symptom-complex, so closely and so repeatedly studied, should still be so variously described and so diversely interpreted.

The student of the subject who seeks for some underlying basis of agreement in the various accounts of the symptom-complex contained in the vast literature of the cerebellum, is as much dismayed by the redundant and elaborate terminology employed as by the various and conflicting senses in which one and the same term is used by different

writers. Indeed, he does not discover that this oppressive and confusing vocabulary conceals a very large measure of agreement as to the facts of observation, until he discards it. Although the impression of confusion and lack of precision obtained from the literature is in part the fruit of a terminology loosely conceived, and still more loosely employed, it has its roots yet deeper in the fact that until recently the state of knowledge did not permit of any precise or accurate formulation of the essential elements of the problem. When so little was known of the nervous processes underlying the co-ordination of movement, and while it was still thought that the cerebellum was concerned with the postural aspects of sensation, a certain vagueness of thought was inevitable. When we recall the severe handicaps under which the pioneer workers laboured in this respect, we cannot but admire the scope and accuracy of their observations.

However, within the past twenty-five years, the classic researches of Sherrington and their development by Graham Brown, Magnus and others, have placed at our disposal a beautiful series of minute analyses of the modes of nervous activity, particularly in respect of the co-ordination of movement and of posture. In consequence, the modern student of the cerebellum has an immeasurable advantage over the investigator of the last century. We can approach the problem with more precise conceptions of the processes concerned in the co-ordination of movement, and with a scientific nomenclature in which to express our conclusions. Moreover, it is now possible to re-survey the earlier observations and, in the light of recently acquired knowledge, to explain much that was formerly obscure.

It is remarkable that, so far, no such re-survey has been attempted, and even the most recent experimental and clinical studies of the cerebellum have not laid this fundamentally important knowledge under contribution. This must be the excuse for the present attempt to examine what we believe to be the fundamental cerebellar defect-symptom from a point of view not yet adequately developed in this connection. It is probable that adherence to what are now obsolete conceptions of muscle tone, and the tendency to regard each particular expression of one and the same disorder of co-ordination as an elementary defect-symptom, are among the principal reasons that recent studies have not carried our knowledge beyond the point at which it stood when Luciani published his observations over thirty years ago. If we take the descriptions by various authorities of the cerebellar symptom-complex in animals and man, and, abandoning the nomen-

clature employed, tabulate in simple descriptive terms the several disorders of muscular contraction and of movement recorded, we can construct such a classification as the following :—

(*I*) *Abnormalities of resting muscle.*—Loss of tone.

(*II*) *Abnormalities of muscular contraction.*—(1) Excessive range and force of contraction ; (2) intermittent and unsteady contraction ; (3) premature relaxation ; (4) extreme readiness of fatigue.

(*III*) *Abnormalities of voluntary movement.* — Faulty functional combination of the muscles engaged in a voluntary movement.

(*IV*) *The effects of voluntary efforts at correction.*—Carried out by a musculature functionally defective in the above senses.

It is apparent at once that this classification cannot be regarded as a final analysis of cerebellar ataxy. For example, the disorders included under heading (*II*) must of necessity impair the perfect functional combination, or synergy, of muscles in a co-ordinated movement, especially in complex purposive movement-combinations. Therefore, asynergia, as the loss of this combination is called, cannot be regarded as an elementary defect-symptom, it must be considered as a manifestation of underlying disorders of muscular contraction. Further, it remains to be determined whether the disorders of muscular contraction are themselves elementary defects, or simply the result of loss of tone. This brings us to the point from which the present study begins, namely, the consideration of the part played by atonia in the production of the cerebellar symptom-complex. As we may readily see from a comparative study [19] of the various descriptions of the cerebellar symptom-complex, a considerable degree of conflict and uncertainty obtains in the use of the term " atonia." It has been variously understood by different writers on the cerebellum, and by none of them has it been used in the sense now adopted by physiologists, namely, as a diminution or loss of postural tone.

Since our conception of muscle tone must govern our use of the term " atonia," a precise definition of what we understand by tone is an essential preliminary to the study we propose. We may say that the modern physiological conception in this respect has scarcely influenced clinical thought as yet. For the clinician, muscle tone is that state of active tension characteristic of living muscle in normal connection with the nervous system, which is felt as a slight but definite resistance to palpation and to passive stretching of the muscle. Conversely, atonia has been taken to mean the loss of this active tension so that the muscle is flaccid, unduly extensible and presents no resistance to passive

stretching. These are the conceptions of tone and atonia employed in the literature on the physiology and symptomatology of the cerebellum. Even with these very restricted interpretations, there is no general agreement as to the importance or constancy of atonia as a cerebellar symptom. Thus, while Luciani [5] and Gordon Holmes [3] accept it as one of the fundamental defect symptoms, Babinski [2] and André-Thomas [1] think it rare and of no importance. Further, the effects of atonia on muscular contraction and on co-ordination of movement are differently interpreted even by those who regard it as an essential symptom. Finally, André-Thomas, who does not employ the term at all, somewhat paradoxically develops a conception of loss of tonic postural nervous activity as a fundamental cerebellar defect symptom, which more truly corresponds to the physiological sense of atonia than any interpretation to be found elsewhere in the literature of the subject.

The sense in which we shall understand muscle tone here is that which Sherrington has so clearly defined [15]. Muscle tone is a purposive reflex reaction, a proprioceptive reflex whose source is in the tonic muscle itself. From his analyses of tone in the decerebrate animal, Sherrington has shown that its physiological purpose is nothing less than the maintenance of posture. Under these conditions, it has a selective incidence in the extensor muscles of the neck, trunk and limbs, that is, in the muscles which maintain the animal in its normal attitude against the force of gravity, the "anti-gravity" muscles. Further, like other reflex reactions employing muscles, it is governed by reciprocal innervation, so that tone is inhibited in the antagonists of the tonic muscles. The hypertonus of the decerebrate animal can therefore be described as "reflex standing." Recently, Magnus has carried out a long series of experimental analyses of the reflex reactions by which tone is regulated and adapted to the varying attitudes imposed upon the animal under experimental conditions. Moreover, he has found that these reactions also obtain, and are the sum total of the reactions occurring in the intact animal in its adoption of all the attitudes of which it is capable. We may conclude, therefore, that muscle tone is the basis of posture and of co-ordinated postural adaptation. This is the only sense in which the term "tone" is employed in this paper, and by atonia we shall understand a loss or impairment of the tonic postural reactions of the normal musculature. If we are to determine the significance of such loss of postural tone in the production of cerebellar ataxy it is by an approach from the physiological standpoint alone that we can hope to reach a satisfactory solution. Therefore, we must begin

with a brief review of the peculiarities of the muscle rendered atonic by experimental procedures in animals. There are two preparations which have been employed by Sherrington in this connection, they are the "decerebrate" and the "spinal" animals. In the former the brain-stem is transected between the levels of the anterior and posterior colliculi. Following this procedure, the extensor muscles of the animal enter into a state of heightened tone. In such an animal preparation a muscle, commonly the extensor of the knee, vasto-crureus, is rendered atonic by "de-afferentation," that is, by section of its afferent nerve supply at the level of the posterior roots. From the reactions of this muscle, the results of loss of postural tone upon reflex reaction can then be investigated. The "spinal" animal is one in which the spinal cord has been transected at some level between the medulla and the lower thoracic segments. The reflex arc of postural tone has its reflex centres in the brain-stem, and therefore such a section cuts the arc and abolishes tone in the muscles innervated from below the level of the transection. However, as we shall see, the atonia produced in this way differs somewhat in degree from that produced by de-afferentation of a muscle in the decerebrate animal.

From a study of the observations of Sherrington under these experimental conditions, the conclusion is reached that *all the component symptoms of cerebellar ataxy are nothing more than manifestations of loss or impairment of postural tone, in other words, atonia is the fundamental cerebellar defect-symptom, all other symptoms being merely particular manifestations of this one defect.*

Chapter II.—The Reactions of the Atonic Muscle.

There can be no doubt that loss of postural tone involves a serious disturbance of balance in the reflex centres concerned in the co-ordination of movement. In the following brief description of the effects of loss of tone upon reflex co-ordination, as Sherrington has revealed them to us, we may hope to reach more accurate conclusions than is possible from clinical observations alone.

(i) *The reflex contraction of the de-afferented muscle.*—Briefly stated, the atonic muscle under these conditions is unduly excitable, contracts more amply than normal and relaxes at once upon cessation of the reflex stimulus. These peculiarities may be considered in detail. The threshold of stimulation, both for excitation and for inhibition, is commonly lowered. Occasionally, however, the threshold may be

normal or even supernormal. The latency of toneless muscle is not altered. The characters of the response are very striking. Contraction develops rapidly and rushes to a maximum, which exceeds the normal, "as though its momentum were less controlled than normal" [11] [13] [14]. Not only is contraction excessive in range, but it is very unsteadily maintained and dies down suddenly as soon as it reaches its height. It does not outlive the exciting stimulus, that is, there is no "after-discharge." A striking result of the last feature is the coarsely clonic or intermittent character of the response to serial stimuli. The normal fusion of component contractions is absent or much impaired [11]. The post-inhibitory exaltation, which normally gives rise to "rebound" contraction, is absent, or is tremulous, ill-maintained and feeble [13]. Fatigue sets in rapidly, so that the response soon wanes, especially in weak contractions [14]. The de-afferented atonic muscle is very susceptible to inhibition, and under this the contraction yields as irregularly as it responds to excitation [14].

(ii) *The reflex contraction of the atonic muscle of the spinal animal.* —The features we have described are equally characteristic of the response obtained from the atonic muscles of the spinal animal, though here they are present to a less extreme degree. In the hind limbs of such a spinal animal several other results of loss of postural tone may be observed. Stimuli, which in the tonic muscle of the decerebrate animal give rise to maintained tonic responses, now evoke alternating movements [13]. Thus, pinching the foot elicits tonic homolateral flexion and crossed extension reflexes in the decerebrate animal, but in the spinal animal clonic alternating movements of flexion and extension, the response opening with homolateral flexion and crossed extension. Similarly, pinching the skin of the perineum in the former results in tonic bilateral extension of the hind limbs, but in free stepping movements in the latter [13]. Finally, the spread of reflex response to regions of musculature remote from that stimulated is much more facile in the spinal than in the decerebrate animal.

(iii) *The nature of the tonic reactions lost to the atonic muscle.*—To understand these phenomena we must have a clear idea of the characters and purpose of the tonic reactions which are absent in the de-afferented muscle and grossly impaired in the muscles of the spinal animal. When a reflex response is elicited in the tonic musculature of the decerebrate animal, the muscular contraction rises rapidly to its height and then declines slowly and often incompletely. The muscle tends

to remain shortened at the new length imposed upon it by the contraction. This "shortening reaction," as Sherrington [11] has called it, is a proprioceptive reflex produced by the initial reflex contraction of the muscle. Like other proprioceptive reactions it is tonic and in effect reinforces the original exteroceptive reflex. Conversely, if the muscle be actively inhibited by some exteroceptive reflex stimulus, the new and increased length reached is maintained by a secondary proprioceptive inhibitory reaction, the "lengthening reaction." The "shortening reaction" is responsible for the tonic "after-discharge" and ensures a steadily maintained contraction in response to serially repeated stimuli. Moreover, after inhibition of tonus, just as after inhibition of active movement, there is a rebound, or post-inhibitory exaltation of tone, which ensures a return to the initial posture broken into by the movement. Thus, these two tonic reactions act as adjuvant and as compensatory reflexes, steadying and maintaining the original contraction and ensuring a return to equilibrium when this has ceased. They give a quality of "plasticity" to muscle tone in virtue of which posture is maintained in the varying attitudes adopted by the living animal.

The effect of the shortening reaction on reflex movement is best and most simply seen in the knee-jerk. As Sherrington [15] and Viets [17] have both shown, there are two elements in the normal knee-jerk, an initial twitch produced by the tap on the tendon, and superimposed upon this a secondary proprioceptive tonic after-discharge or shortening reaction. In a myogram it is seen as a gradual and incomplete fall of the limb. This shortening reaction is entirely absent in the knee-jerk obtained from the atonic muscle, in which the relaxation is sudden and complete.

(iv) *Atonia and reflex co-ordination.*—If the reflex contraction of a muscle is disordered by loss of tone in the manner we have described, it follows that the reflex co-ordination of movement must suffer even more. In the case of the simple reflex elicited by a chosen stimulus we have two groups of muscles involved, agonists and antagonists. Under reciprocal innervation, when the former group is excited to contract, the latter is inhibited. The atonic muscle behaves as irregularly under inhibition as when contracting. So that side by side with excessive amplitude of movement, abruptness of onset and cessation, fatigability, irregular yielding to inhibition, and, in the serial responses, a clonic intermission of the contraction wave, we have in addition a negative side of all these defects present in the antagonist group. But the simple reflex, employing reciprocal innervation in an otherwise quiescent muscu-

lature, is an abstraction, an artificial state of affairs never reproduced during life [14]. Here conflicting stimuli pour continuously upon the surface of the organism and compete for possession of the different muscular mechanisms. At a given moment, therefore, any reflex centre is under a twofold influence, one tending to excite the muscles governed by it to contraction, the other to inhibit them, or, at least, to employ them in a reaction of different character and purpose. In the normal organism the result is not chaos but perfect co-ordination. Sherrington has called the process by which this is achieved *double reciprocal innervation* [12] [14]. When two conflicting stimuli struggle for possession of a final common path, the resulting reaction depends upon their relative intensities. If there be considerable disparity, one displaces the other which is suppresed, but if their intensities be relatively equal the result is an algebraical summation of the effects. The muscle contracts, but less powerfully than it would have done under a single exciting stimulus, and its antagonist is inhibited, but less completely than it would otherwise have been. Therefore, each muscle is under a twofold influence, it is at once excited and inhibited. In the agonist the former effect predominates, in the antagonist the latter. Therefore, both muscles of an antagonistic pair are in movement or in tonic contraction simultaneously. But under double reciprocal innervation, phasic or tonic contraction does not increase simultaneously in both. As it waxes in one it wanes in the other, except when both start from quiescence, when contraction is stronger in the agonist than in the antagonist. This combination of excitation and inhibition is a fundamental factor in the grading of reflex contraction.

The atonic muscle with its loss of balance and stability in reflex reaction must, under these complex conditions, give rise to still more marked disorders of co-ordination. Moreover, in the normal individual voluntary movement involves the breaking up of the simple reflex movements into combinations and sequences of almost infinite variety and complexity [4]. Vitiating all these complex activities in direct proportion to their complexity is the uncontrolled, abrupt and unsteady character of reflex response occurring in an atonic musculature. We see that tone is intimately interwoven with movement in the processes of perfect co-ordination. To use Jackson's expressive phrase, there is "a perfect co-operation of antagonism" between the phasic and tonic elements in the co-ordination of movement. How intimate this association must be, and how essential its maintenance, is apparent when we remember that all our movements demand the finest postural adapta-

tion. The spinal animal can step, but it cannot walk because it is unable to stand. The lecturer who writes on a blackboard maintains his arm in the extended and elevated position while his forearm and hand muscles execute the movements of writing. This postural adaptation is achieved by the reflex modification of tone. If his muscles be atonic, he has consciously and by voluntary direction to hold the arm extended by a series of movements each of which subsides immediately unless it be repeated. The contractions underlying this voluntary replacement of what is normally a subconscious reflex process will have all the characters of an atonic musculature; they will be excessive in range and force, subject to premature relaxation, to fatigue and to intercurrent inhibition. In the maintenance of the erect posture and in movements of locomotion where postural reactions play an even more obviously important part, it is inevitable that there should be the gravest disorders of co-ordination, and therefore of the maintenance of equilibrium.

(v) *The spinal component of tone.*—We cannot leave the discussion of atonia without reference to the significance of the spinal component of tone. Tone is observed in the muscles of the spinal animal after recovery from "spinal shock." For various reasons it is more readily studied in the dog than in the cat. It is never observed in the spinal monkey [9]. The immediate result of spinal section in the lower thoracic region in the cat is to produce a flaccid paraplegia. A brisk knee-jerk is obtainable immediately after recovery from the anæsthetic. By the next day there is usually appreciable tone in the hip flexors, and very shortly in the adductors of the thigh. At first the extensors remain flaccid, but, even in these muscles, in four or five days transient accesses of tone may be observed. In every instance, and whatever group of muscles is in question, this spinal tone is peculiarly fugitive and uncertain of development. It is liable to sudden and complete lapses. A definite shortening reaction is rarely obtained, and, in short, spinal tone lacks plasticity and is most ineffectual as a postural reflex. Sherrington [11] finds that the spinal dog can be made to stand for a few seconds, but here, as in the cat, tone is apt to disappear suddenly and the animal collapses. Therefore, while passive movement may indicate the presence of tone in the muscles under these conditions, yet in the strict sense postural tone may be considered to be absent. This is an observation not without significance in relation to the atonia of cerebellar lesions, and will be discussed later.

While it is found that in the decerebrate animal postural tone has a selective incidence in the extensor group of muscles and is not readily discoverable in flexors, we must remember that decerebrate rigidity is an exaggerated tone. Since reciprocal innervation obtains for tone, it follows that in the decerebrate animal flexor tone is unduly inhibited. We might expect the distribution of tone in the intact animal to show less inequality in this respect, though all the evidence indicates that the extensors form the more important group where tone is concerned. This is readily comprehensible when we recall that they are responsible for the maintenance of the normal attitude of standing. The observations of Magnus are of great interest in this connection [6] [7]. He finds, in confirmation of the earlier work of Thiele [16], that decerebrate rigidity does not appear until the plane of transection of the brain-stem passes caudal to the anterior colliculi. An animal (rabbit, cat, dog or ape) in which the plane of transection passes immediately anterior to the mid-brain does not show decerebrate rigidity, its tone has what Magnus calls a "normal distribution." Such a mid-brain animal can not only stand, it can walk and jump and, if overturned, can right itself again. All its postural mechanisms are intact. In other words, the reflex arcs necessary to the maintenance of tone, and the reflex mechanisms by which it is regulated in the interests of co-ordination, are all intact.

When the transection lies sufficiently caudal to produce decerebrate rigidity some of these regulating mechanisms have been removed and simple tone, released from control, remains. In the de-afferented muscle of the decerebrate animal and in the atonia of the spinal animal the arcs concerned in the maintenance and the regulation of tone have both been cut out.

(vi) *The muscle rendered atonic experimentally, compared with the "ataxic" muscle of cerebellar lesions.*—At first sight there might appear to be one grave objection to the comparison of such relatively artificial preparations as muscles rendered atonic by the experimental procedures indicated with the "ataxic" muscle of a case of cerebellar disease or injury. It is true that the de-afferented muscle of the decerebrate preparation has been deprived of both sensory and non-sensory afferent fibres, while the affected muscle in cerebellar disease has its sensory path intact. But in the decerebrate animal the sensory path has already been interrupted by the removal of the cerebral hemispheres, and yet tone and all tonic reactions are intact. Therefore the absence of sensory afferent fibres does not introduce a complicating factor.

The maintenance of tone is a function of the non-sensory afferent group, and it is to the physiological disturbances in the central connections of this group that the "ataxy" of cerebellar lesions is due.

The condition of the muscles of the spinal animal is somewhat different. Here also the sensory path is destroyed together with the long reflex arc of postural tone. The local spinal arc, however, is intact and hence the purely spinal, phasic component of co-ordination remains.

In these circumstances, some tone, it is true, does return to the muscles, but it has little plasticity or postural value, is inconstant, liable to sudden complete lapses and is not comparable with the extensor muscle tone of the decerebrate preparation. Hence the relatively atonic muscle of the spinal animal shows the defects seen in the de-afferented muscle, though to a less complete degree.

From another point of view there appears at first sight a difference between the activities of the atonic muscle in the experimental animal and those of the "ataxic" muscle of the subject of a cerebellar lesion. The former is capable of reflex responses alone, while the ataxy of cerebellar lesions is manifested in voluntary activities. From what we know of the functions of the cerebral motor cortex (Leyton and Sherrington) it appears probable that the actual processes of co-ordination are a function of the lower reflex levels of the nervous system, and voluntary movement is but the activation of these reflex mechanisms for the production of an infinite variety of complex movement combinations and sequences. The large and relatively simple motor reactions of the decerebrate or spinal animal are the material from which, by a process of analysis and synthesis, the cerebral cortex elaborates the skilled and finely adapted movements of the normal individual. In the subject of a cerebellar lesion, just as in the experimental animals we have been considering, it is the activity of the reflex levels which is interfered with, so that we may conclude that the presence of volitional activity in the case of purely cerebellar lesions does not invalidate the comparison we are about to make between the disorders of co-ordination produced by loss of postural tone aud the motor disorders which we include under the heading of cerebellar ataxy.

Chapter III.—The Cerebellar Symptom-Complex.

We are now in a position to review the cerebellar symptom-complex in the light of the experimental observations we have described. So apparent is the identity of many of the symptoms of cerebellar lesions with the characteristic reactions of the atonic muscle that no lengthy consideration is required If we take the disorders included under heading (*II*) of the table on p. 541, we see that they are all disorders which occur when postural tone is abolished. We have already discussed the clinical conception of atonia. Its limitations are sufficient to explain how it is that " atonia " has not been regarded as of more importance by the clinician. It is true that Luciani concluded that atonia, asthenia and astasia were but different expressions of a single defect, and that his definition of tone includes phenomena which subsequent experimental research has shown to be manifestations of loss of postural tone. We cannot but regret that physiological knowledge of the processes concerned in the co-ordination of movement was not more advanced at the period of Luciani's investigation. For so accurate are his observations and so penetrating his conclusions that could he have reviewed them from the standpoint of modern knowledge of the physiology of the nervous system, we might well have been within sight of a satisfactory theory of cerebellar functions.

There are authorities who deny the occurrence of atonia, in the clinical sense, as a cerebellar symptom, or who believe that muscle tone may be restored before perfect co-ordination returns. From what has been said of the spinal component of tone we can readily understand that some recovery from the profound muscular flaccidity of a recent acute cerebellar lesion may well occur while ataxy is still observed, or that in other instances no complete flaccidity on palpation or on stretching may be seen. Yet this does not necessarily indicate that postural tone is present in the cases in question. As we have seen, the tests employed for the estimation of tone, as this is understood clinically, are not competent to decide this point. However, if we accept the hypothesis now put forward that cerebellar ataxy is itself the direct and inevitable result of postural atonia, we shall no longer be restricted to these inadequate standards, but shall have every test for cerebellar ataxy as a criterion of postural tone.

While it is scarcely necessary to go *seriatim* through all the disorders of voluntary movement observed in the subjects of cerebellar lesions, we may briefly take one or two of the most characteristic ones

and consider them as possible results of postural atonia. Let us take, for example, the ready fatigue, the excessive range of movement ("dysmetria") and the oscillations or jerky variations of sustained movements ("discontinuity of movement"). They are found to be, without exception, inevitable results of loss of postural tone.

As Luciani pointed out, we cannot exclude the complicating effect of voluntary efforts at compensation and correction, themselves carried out in an atonic musculature. They must aggravate the disorder of movement in voluntary motor activities, but in the ultimate analysis atonia or loss of postural tone is an adequate explanation of all the manifestations of cerebellar ataxy. Finally, the graphic records of the knee-jerk obtained in cases of acute cerebellar lesion by Gordon Holmes show an ample twitch contraction without shortening reaction, a typical atonic reflex response. André-Thomas has also described this type of knee-jerk as characteristic of cerebellar lesions.

A further point remains for consideration. Luciani observed that after ablation of the cerebellum, and at a stage when it could not stand, a dog thrown into water was able to swim without any defect of co-ordination. From this he concluded that inco-ordination, in the strict sense of the term, did not occur as a result of cerebellar lesions. The essential defect was an asthenia in virtue of which the animal was not able to support its weight, and the water, by overcoming this difficulty, rendered perfect harmony of movement possible. This remarkable observation does not appear to have attracted the attention it merits. In the light of the present hypothesis it assumes considerable significance, and may be taken to afford some confirmation of the point of view presented in this paper. Movement in such a resistant medium as water must, though imperfectly, control many of the most characteristic results of loss of postural tone; for example, the suddenness of contraction and of relaxation, the excessive amplitude and force of contraction, and the oscillations resulting from loss of postural fixation. Indeed, we could scarcely devise an experiment more calculated to demonstrate the fundamentally important rôle of atonia in the production of cerebellar ataxy.

In conclusion, the hypothesis which has been advanced may, with its corollaries, be summarized as follows: Hughlings Jackson's view that two components, "tonic and clonic," were concerned in the perfect co-ordination of movement has been given experimental verification by the researches of Sherrington. We now speak of tonic and phasic elements in co-ordination. The former, the basis of which is muscle

tone, is concerned with the maintenance and regulation of posture, and between the two elements there is, in Jackson's words, "a perfect co-operation of antagonism." The essential result of a cerebellar lesion is the impairment or abolition of tone, and therefore of all tonic or postural reactions. It is strictly accurate to speak of this as atonia, and in this sense alone we have employed the term. Therefore, in saying that atonia is the essential cerebellar defect-symptom, we express the loss of one of the two essential elements in motor co-ordination. The diverse and conflicting analyses of the cerebellar symptom-complex to be found in the literature describe a large number of so-called elementary defect-symptoms. However, as soon as we adopt the physiological conception of muscle tone and abandon the redundant and empirical nomenclature now employed in this connection, we find that the symptoms which all observers have agreed in describing are nothing but various manifestations of loss of postural tone. In this way we have no difficulty in harmonizing the facts of observation as recorded by different authorities with the hypothesis now put forward.

In this connection it may not be without interest to consider whether what Jackson called a "corresponding opposite" to the combination of loss of postural tone with retention of voluntary power ever occurs in man under the conditions of disease; namely, a state of affairs in which with loss of voluntary power postural tone is preserved. The present writer has suggested elsewhere [18] that such a dissociation of function does occur in spastic hemiplegia where the paralysed limbs show involuntary "associated movements" when the normal limbs are voluntarily put into action. The postural reactions which normally accompany any movement, especially if this be forceful or widespread, are bilateral. In their papers Magnus and de Kleijn have given us many examples of widespread postural reaction, and Sherrington has described the postural accompaniment of the scratch reflex. In hemiplegia the loss of voluntary control over the limbs of the paralysed side cannot result in the suppression of such reactions on that side, but in their "release." Under these conditions we may expect them to develop in an exaggerated form, but lacking in that refined character which they normally possess. It is reasonable to suppose that the associated movements of the paralysed limbs in hemiplegia are manifestations of this nature. If this be so, we have a very familiar "corresponding opposite" to cerebellar ataxy. In both instances the falling out of one of the component elements in the process of co-ordination results in gross impairment of the factors remaining,

whatever be the physiological rank of the lost factor. Thus voluntary movement is disordered from loss of postural tone and postural reactions suffer when voluntary control is withdrawn.

Chapter IV.—Some Theoretical Conclusions.

If we accept this hypothesis as the most satisfactory generalization of the facts of observation, we can readily see how it is that the older analyses have failed to provide the basis for any theory of cerebellar functions. As we have indicated, the most concise analysis is that of Luciani's, but at the time it was formulated so little was known of the nervous processes underlying the maintenance and regulation of muscle tone that it was not possible for him to achieve a final solution of this fascinating and difficult problem. We may now pass to consider very briefly whether the point of view we have adopted in the present paper provides a more promising vantage ground for an attack upon the physiological problems presented by the cerebellum. It would be easy to conclude at once that the cerebellum is the organ for the regulation and integration of all the elements concerned in the maintenance and regulation of posture. Unfortunately, the matter is by no means so simple, and such a view does not take into account some recently recorded observations of Magnus. Our first difficulty is to explain how a defect lesion of the cerebellum produces loss of postural tone. When we compare the results of ablation of this organ in the intact and in the decerebrate animals a striking difference appears. Whereas all observers are agreed that an acute and purely cerebellar lesion in man, or the ablation of the organ in an animal with an otherwise intact nervous system, produce the characteristic cerebellar ataxy, yet, as Sherrington was the first to point out many years ago [10], in the decerebrate animal the rigidity is not abolished by subsequent removal of the cerebellum, nor conversely does ablation of the cerebellum prevent the development of rigidity upon subsequent decerebration. Further, Magnus [6] [7] [8] finds that not only does rigidity persist after ablation of the cerebellum, but all the labyrinthine reflexes which normally play so fundamental a part in the co-ordination and regulation of muscle tone are intact. In other words, the reflex paths concerned in the maintenance and regulation of tone do not pass through the cerebellum. Magnus concludes that we cannot regard the cerebellum simply as the central nervous organ of the labyrinth, and so far as the decerebrate animal is concerned it seems as though we cannot even

regard the cerebellum as essential to the maintenance and regulation of posture. *In short, in the intact animal, the cerebellum appears essential to the maintenance and regulation of postural tone, but in the animal deprived of its cerebral hemispheres it is not essential to either process, as far as either is carried out under these conditions.* The whole question of tone, and the various ways in which it may be affected by different lesions of the nervous system, as we have discussed them, may be simply expressed in the following diagrams. Here the lesion is indicated by a heavy line and the resulting state of postural tone is tabulated in the legend.

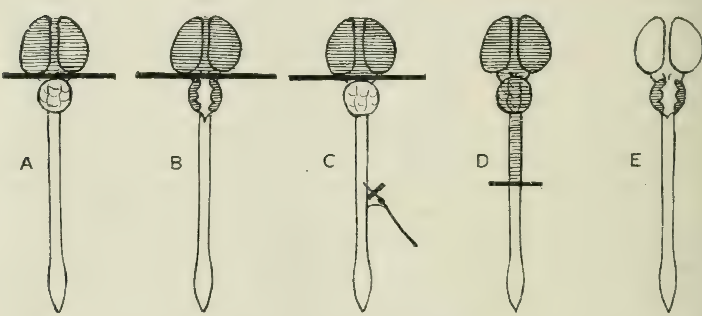

FIG. 1.—A, Decerebrate animal with intact postural tone; B, decerebrate animal with ablation of cerebellum and intact postural tone; C, decerebrate animal with posterior root section and loss of postural tone in "de-afferented" muscles; D, spinal animal with loss of postural tone; E, ablation of cerebellum, central nervous system otherwise intact; loss of postural tone. Heavy lines indicate the situation of the lesion. The portions of the nervous system out of action are shaded. Ablation of the cerebellum, or gross lesions in man, are indicated by shading of cut cerebellar peduncles.

The explanation of this apparent anomaly is probably to be found in certain limitations inherent in these experimental observations. The decerebrate animal is a reflex machine devoid of all volition. Its reflex reactions, although perfectly co-ordinated, consist in relatively simple mass movements, for which the reflex centres situated in the brain-stem are adequate. The voluntary motor activities of the intact animal, and still more those of man, display innumerable variations and elaborations of these simple movements. From their studies of the responses of the anthropoid motor cortex, Leyton and Sherrington [4] draw the inference that it is the function of this region of the cerebral cortex so to influence the lower level reflex mechanisms which are directly con-

cerned in the co-ordination of movement that the skilled, isolated movements of the individual are produced. The cortex by its activity analyses the large and simple reflex movement-complexes into their fractional elements, and synthesizes these into combinations and sequences of almost infinite variety, which make up the normal purposive movements of the individual. We have seen that there are postural and phasic factors concerned in these processes. May we not suggest that the cerebellum is the organ through which the cerebral motor cortex influences postural activities and regulates posture in the interests of co-ordinated purposive movement? It may be the subordinate mechanism employed by the cerebral cortex in the refined correlation of posture with movement.

It has been the custom to think of the cerebellum as a physiological as well as an anatomical entity, though like any other anatomical subdivision of the brain it can have no physiological meaning apart from the rest of the nervous system. That we should continue so to regard it is probably a potent factor in our inability to form any convincing theory of its functions, and a relic of the period when the localization of function in the brain was envisaged with an almost geographical precision. Once we have assimilated the conception of postural and phasic components of movement, and have come to regard the cerebral motor cortex not as itself directly concerned in the processes of co-ordination, but as playing upon the lower level mechanisms which are engaged in this function, and as integrating the postural with the phasic elements of motor activity, it becomes somewhat easier to see why the results of cerebellar lesions should be so different in the intact and the decerebrate animals. In all probability, it is in the light of its functional relationships with the cerebral cortex that we shall ultimately gain an insight into the functions of the cerebellum. Certainly, the parallel development of cerebral cortex and neo-cerebellum, as revealed by comparative anatomy, points the way to such a conclusion.

REFERENCES.

[1] ANDRÉ-THOMAS. "Cerebellar Functions," Journ. Nerv. Dis. Publishing Company, New York, 1912.

[2] BABINSKI, J., et TOURNAY, A. "Les symptômes des maladies du cervelet et leur signification," *Trans. XVIIth Internat. Cong. Med.*, London, Section XI, Neuropathology, **1**, 1.

[3] HOLMES, GORDON. "The Symptoms of Acute Cerebellar Injuries due to Gunshot Injuries," *Brain*, 1918, **40**, 461.

[4] LUCIANI, L. "Human Physiology," London, 1915, **3**, 419.

[5] Leyton, A. S. F., and Sherrington, C. S. "Observations on the Excitable Cortex of the Chimpanzee, Orang-utan, and Gorilla," *Quart. Journ. Exper. Physiol.*, 1917, **11**, 135.

[6] Magnus, R. "Welche Teile des Zentralnervensystems müssen für das Zustandekommen der tonischen Hals- und Labyrinthreflexe auf die Körpermuskulatur vorhanden sein?" *Arch. f. d. ges. Physiol.*, 1914, **159**, 224.

[7] *Idem.* "Beiträge zum Problem der Körperstellung: I. Mitteil. Stellreflexe beim Zwischenhirn- und Mittelhirn-Kaninchen," *Arch. f. d. ges. Physiol.*, 1916, **163**, 405.

[8] Magnus, R., und de Kleijn, A. "Ueber die Unabhängigkeit der Labyrinthreflexe vom Kleinhirn und über die Lage der Zentren für die Labyrinthreflexe in Hirnstamm," *Arch. f. d. ges. Physiol.*, 1920, **178**, 124.

[9] Sherrington, C. S. "Experiments in Examination of the Peripheral Distribution of the Fibres of the Posterior Roots of some Spinal Neives" (Croonian Lect., R.S., 1897), *Phil. Trans. Roy. Soc.*, 1898, **158**, B, 45.

[10] *Idem.* "Decerebrate Rigidity, and Reflex Co-ordination of Movements," *Journ. Physiol.*, 1898, **22**, 319.

[11] *Idem.* "On Plastic Tonus and Proprioceptive Reflexes," *Quart. Journ. Exper. Physiol.*, 1909, **2**, 109.

[12] *Idem.* "Reciprocal Innervation of Antagonistic Muscles; Fourteenth Note—On Double Reciprocal Innervation," *Proc. Roy. Soc.*, 1909, **81**, B, 249.

[13] *Idem.* "Flexion Reflex of the Limb, Crossed Extension Reflex, and Reflex Stepping and Standing," *Journ. Physiol.*, 1910, **40**, 28.

[14] *Idem.* "Reflex Inhibition as a Factor in the Co-ordination of Movements and Postures," *Quart. Journ. Exper. Physiol.*, 1913, **6**, 251.

[15] *Idem.* "Postural Activity of Muscle and Nerve," *Brain*, 1915, **38**, 191.

[16] Thiele, F. H. "On the Efferent Relationship of the Optic Thalamus and Deiters' Nucleus to the Spinal Cord, &c.," *Journ. Physiol.*, 1905, **32**, 358.

[17] Viets, H. "Relation of the Knee-jerk and Patellar Clonus to Muscle Tonus," *Brain*, 1920, **43**, 269.

[18] Walshe, F. M. R. "On the Genesis and Physiological Significance of Spasticity and other Disorders of Motor Innervation, with a Consideration of the Functional Relationships of the Pyramidal System," *Brain*, 1919, **42**, 1.

[19] *Idem.* "The Cerebellar Symptom-complex and its Significance," *Medical Science*, 1921, **5**, 134.

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Persistent Pain in Lesions of the Peripheral and Central Nervous System.

By WILFRED HARRIS.

PERSISTENT pain, which may be defined as acute bodily discomfort, varying in degree from a sensation of soreness or aching to one of intense and intolerable agony of torment, may be due to a variety of diseases affecting primarily other structures and tissues than the nervous system. Thus, carcinoma naturally occurs to us, or other malignant growth, as a probable cause of any deep-seated pain, persistent in character and lasting for weeks and months, more especially if accompanied by progressive emaciation.

The appreciation of any sensation of pain due to visceral or bone disease necessarily involves the agency of the nervous system in the transference of the sensory impulses from periphery to thalamus and cerebral cortex; but I am limiting my remarks as far as possible to diseases involving the nervous system itself, and shall omit discussion of the referred pains of visceral disease, such as dental neuralgias, errors of refraction, angina, the colics, sinus suppuration, abscesses, acute inflammations and the like.

For the sake of convenience I have in the following classification arranged the causes of persistent pain in diseases of the nervous system in five classes, progressing from periphery to centre, or from nerve-ending to cerebral cortex.

Persistent Pain in Lesions of the Peripheral and Central Nervous System.

(1) *Peripheral due to inclusion of nerve-endings in scar.*
  (*a*) e.g., Neuro-fibrositis, traumatic or rheumatic; adiposis dolorosa.
  (*b*) Due to septic or to rheumatic terminal trigeminal neuritis:—
    (i) Chronic paroxysmal trigeminal neuralgia (Fothergill's disease, tic douloureux).
    (ii) Chronic neuralgia of upper or lower jaw.
  (*c*) Geniculate neuralgia.
  (*d*) Glossopharyngeal neuralgia.

(2) *Disease involving nerve trunks.*
  (*a*) Supraorbital neuralgia.
  (*b*) Multiple neuritis.
  (*c*) Brachial or sciatic perineuritis.
  (*d*) Tumours or gummatous neuritis:—
    (i) Trigeminal.
    (ii) Spinal, e.g., neurofibromatosis.
  (*e*) Causalgia.
  (*f*) Cervical or first rib pressure.

(3) *Lesions of posterior root ganglia or posterior roots.*
  (*a*) Post-herpetic neuralgia:—
    (i) Trigeminal.
    (ii) Spinal.
  (*b*) Tabetic neuralgia.
  (*c*) Other root scleroses.

(4) *Central sclerosis of fillet or thalamus.*
  Intra-medullary growths.
  Syringomyelia and syringobulbia.

(5) *Psychalgias.*

Neurofibrositis.—The pains of chronic or acute fibrositis of the lumbar or dorsal region, often known as lumbago and muscular rheumatism, are doubtless only too well known to many of us, though common as the affection is, its pathology is largely a matter of conjecture. Violent or, and especially, sudden muscular action is the exciting cause in a considerable number of cases: the sudden onset of intense pain immediately after a heavy muscular strain, such as a slip or fall when carrying a heavy weight, the pain relieved by

lying down or sitting in a deep chair and aggravated immediately on movement, suggest rupture of muscle or tendinous fibres involving sensory nerve filaments, as the cause. In the majority the pain disappears after a few days or weeks, while occasionally the disorder persists for years.

Recently I was consulted by a farmer, a powerfully built muscular man, who some ten years ago slipped and fell when carrying a sack of wheat weighing about 260 lb. He was immediately seized with acute pain across the lumbar region, and more especially in the neighbourhood of the right posterior iliac spine. He has been subject to this pain ever since off and on, worse on movement or during heavy lifting, and now and again he is subject to exacerbations so severe he can scarcely move or dress himself. When I saw him during such an attack, his spine was flexed laterally towards the right, and there was a small circumscribed area of great tenderness on deep pressure over the right posterior iliac spine.

Careful marking of the site of chief tenderness, followed by injection of a few minims of 90 per cent. alcohol deep into the tender area, completely relieved his pain within fifteen or twenty seconds, so that he was able to stand up straight, the lateral flexion of the spine having now disappeared, and he walked away in comfort.

Acupuncture has long been recommended since Sydney Ringer's time for similar conditions, but if one or more definite areas of tenderness on deep pressure are discovered, I consider that alcohol puncture is far more valuable.

Traumatic neuro-fibrositis may involve larger nerve trunks, not nerve filaments only, and sciatic perineuritis is a fairly common early sequel to fibrositis of the lumbar region, whether this is of rheumatic origin or due to a fall or other injury. Here again the pain may continue for months or even years. Sciatic perineuritis as a sequel of muscle strain or fall is particularly amenable to treatment by massive saline injections into the nerve just below the notch, the probable site of the nerve sheath damage. Alcohol injection of the sciatic must, of course, never be attempted, owing to the certain result of paralysis of the foot which would ensue, but saline injection of 50 or 60 c.c. preceded by 2 c.c. of 2 per cent. novocain into the nerve often cure the sciatica immediately. This treatment, though often valuable, is less certainly successful in the rheumatic sciaticas, possibly owing to the large area of the nerve which may be the seat of trouble. In both the rheumatic and the traumatic sciaticas there

are frequently spots of deep tenderness with aching pain either in the neighbourhood of the trochanter or between the notch and iliac crest, which may keep up the discomfort and pain after the actual sciatica has been relieved. These are probably local areas of neurofibrositis in the glutei or erector spinæ, and may be mostly successfully injected deeply with alcohol, after the nerve itself has been treated with the saline injections. Probably the saline injections relieve by breaking apart laterally adhesions of the nerve sheath, as the sciatic nerve is a loosely built nerve and readily takes the fluid, swelling up like an egg at the site of injection (as may be seen by injecting the nerve after exposing it on the post-mortem table).

Paralysis of the sciatic I have never seen as a result of violent muscular action, but in the region of the brachial plexus local paralysis from involvement of the posterior thoracic, long thoracic, or circumflex nerves may occur. As with sciatica, so *brachial perineuritis* may occur from muscular overstrain, or rheumatism and other toxic causes, such as pyorrhœa. Usually the posterior cord and musculo-spiral nerve suffer most. As in the lumbar region, so also in the neighbourhood of the scapula and sometimes in the forearm, areas of deep tenderness may be the sole cause of chronic wearing pain, or may accompany a more extensive brachial neuritis. Alcohol puncture of such tender spots often has most brilliant results, chronic pain that may have persisted for many months or years being relieved instantaneously. The spots must be carefully located and marked, and a fine needle, hypodermic size, is usually long enough, plunged vertically into the spot down to the level of the rib or scapula. Care must of course be taken that the needle does not pass between the ribs and pierce the pleura.

Bruising of the scalp by an injury to the vertex from a blow against the lintel of a door, or fall of a heavy weight, is sometimes followed by persistent headache, varying in intensity, or periodic, and associated with a local area of tenderness on pressure. These cases also may be relieved by the same treatment. This form of headache must be distinguished from migrainous neuralgia, in which the periodic headache is unilateral and often associated with intense pain and tenderness on pressure on one temple. Alcohol injection in these cases is quite useless, as is only to be expected, the origin of the pain being probably central and not peripheral.

Chronic paroxysmal trigeminal neuralgia is certainly of peripheral origin, and is probably due to septic neuritis of nerve filaments in the maxilla or mandible. Scarcely ever does this disease affect the

upper division and supraorbital alone, but it may be involved along with the second division.

John Fothergill's original description nearly one hundred and fifty years ago of the painful attacks is scarcely to be improved on to-day, though his view of its pathology, thinking that it must be cancerous, because of the long-standing pain, is now discarded. Many causes appear to contribute to its appearance. Heredity of the disease I have met with in about 1 per cent. of my cases. Strong emotion may precipitate an attack. Injury and blows on the jaw are a not unfrequent cause, as may also be exposure to severe chill, as in motoring or driving; a septic antrum has preceded typical tic douloureux of the second division too often to be a coincidence. I find the disease twice as frequent in women as in men, a point also noted by Fothergill, though he saw only sixteen cases in his practice (or business, as he calls it). A curious point my statistics show is that the disease is twice as frequent on the right side as the left, and commoner in the upper than the lower jaw, though both are often affected. The larger incidence on the right side may be due to better use of the toothbrush on the left side of the mouth, as would be natural in right-handed people, according to Mr. Warwick James. Bilateral trigeminal neuralgia is comparatively rare. I have met with it perhaps thirty times only among several hundreds of cases. The disease rarely disappears spontaneously when established (I have known this occur only once, in the father of a sufferer from this disease, who was my patient). Her father suffered for twenty-five years until he was aged 90, but was free for the last fifteen years of his life, living to 105. In the early stages, however, long periods of remission may occur; I have known thirty years elapse between the first and second attacks, and intervals of one to two years are common. Sometimes a certain periodicity is seen, as for a few weeks to three months every autumn or winter. As the years pass usually the intervals of freedom get shorter. In a few, there is practically no remission, agonizing pain inevitably following every movement of the face, as in eating or in washing. One of my patients, a lady, had been unable to wash the right side of her face for twenty years. Often the lightest touch of a hair or draught of air will start a paroxysm, though at other times nothing may provoke the pain. During the painful bouts the prick of a pin usually seems more acute on the affected side, though this is a temporary hyperæsthesia only, and is not present between the attacks.

Fothergill, 150 years ago, thought hemlock pushed to toxic doses was a cure for this complaint, but our experience nowadays is that drugs comparatively rarely are of service, and that real relief is obtained only by a solution of continuity of the offending nerve trunk. Practically the choice, in severe cases of pain, is between gasserectomy or division of its sensory root, both severe operations, on the one hand, and alcohol injection of the nerve trunks at their deep foramina, or injection of the ganglion itself. For bilateral cases of this neuralgia the gasserectomy operation is not permissible, owing to the jaw drop that would ensue, though bilateral destruction of the ganglion by injection may be done, as the motor root then recovers, though the sensory ganglion cells are permanently destroyed. A curious point I have often noticed when injecting the foramen ovale and ganglion is that the ophthalmic or inner portion of the ganglion goes totally anæsthetic before the second division, and it may be extremely difficult at times to get total anæsthesia of the cheek when the forehead and eyeball remain perfectly anæsthetic. I have no satisfactory reason to account for this.

Another puzzle that occasionally, though fortunately rarely arises, is the partial recurrence of sensation on the chin and remainder of the third division, when the first and second divisions remain totally anæsthetic. With this reappearance of sensibility, pain may return, and that this is not necessarily the result of the faulty injection is proved by the fact that gasserectomy and division of the sensory root behind the ganglion may not alter the conditions. One is tempted then to call the persistent pain a psychalgia, but of this I am not convinced, as there is no doubt true sensation on the lower lip and chin. Gasserectomies, of course, like other operations, may sometimes fail through being incomplete; I have seen several such cases where pain has returned after about ten years, with reappearance of cutaneous sensibility, yet I do not think that is the explanation in the few cases above referred to, though no such case that I am aware of has come to autopsy.

Another form of *persistent trigeminal pain* that I have seen many instances of is in my experience peculiar to young women. It is continuous, not paroxysmal, though it may vary in severity, and it affects either the upper or the lower jaw. It is not provoked by eating, laughing, washing or other movements of the face as is true tic douloureux. It is more difficult to relieve by alcohol injection than spasmodic tic douloureux, inasmuch as total anæsthesia is

necessary to abolish the pain, and with commencing regeneration of the nerve the pain recurs. In tic douloureux, however, in a large majority a medium anæsthesia from injection may be sufficient to abolish the pain, in some cases for many years; in one of my early cases there has been no recurrence $12\frac{1}{2}$ years after injection of the foramen-ovale, though only light anæsthesia now remains.

This lady had previously suffered from typical tic for twenty years, section of the inferior dental nerve having given only two years' relief. In her case the neuralgia commenced at 17, and I have seen it commence at 17 in two other cases and once at 16, many in the twenties, but most commonly about the age of 50. On the other hand the type of persistent neuralgia of upper or lower jaw previously described I have met with only in women of 15 to 35. Its cause I am very uncertain about, unless it is a chronic osteitis of the jaw. Its limitation to women I do not understand, as the sufferers I have met with have not been notably of the neurotic type. Moreover, the true tic douloureux is undoubtedly much more frequent in women than in men, about two to one. These cases are more difficult to treat than true trigeminal neuralgia, as nothing short of total nerve destruction cures the pain.

Supra-orbital neuralgia.—Persistently recurrent supra-orbital neuralgia is met with in both sexes, though oftener in women. I assume that migrainous and true trigeminal neuralgia, and the peripheral causes such as errors of refraction, frontal sinusitis, frontal herpes, antral abscess and dental neuralgias have been excluded. Sometimes paroxysmal supra-orbital neuralgia occurs daily, coming on about the same time, perhaps 10 or 11 a.m., and lasting until 5 p.m. I have seen this type follow influenza several times. Usually the pain is limited to the supra-orbital nerve, but it may involve the whole of the ophthalmic branch. In a recent case, a woman of 47, pain began four years ago, lasting for eight hours daily from January to June. Next year and the year after a similar repetition occurred. A year ago she had a more severe attack and since then has had pain daily from 12 noon to 5 p.m., never a day free. The pain is situated in the forehead and left side of head as far back as the coronal suture, and extends along the left side of the ridge of the nose. Unlike trigeminal neuralgia the pain is not evoked by talking, eating, or other movements of the face, or by rubbing. Supra-orbital injection gave little or no relief, though deep anæsthesia was produced. Injection of the Gasserian ganglion was then done, the anæsthesia of the first division being total

for an hour or more, but subsequently wore off partially. The neuralgia disappeared completely for several days, and then reappeared in a much attenuated form. Very probably total destruction of the ganglion would have produced a complete cure of the neuralgia.

Perhaps the majority of paroxysmal supra-orbital neuralgias are migrainous in type, for which alcohol injection is of little or no use. In some subjects this migrainous periodic neuralgia is limited to the temple.

Only comparatively rarely does true trigeminal neuralgia invade the first division of the fifth nerve, that is to say a paroxysmal neuralgia, sudden and intense, and brought on by movements of the face, rubbing of the skin, or even a draught. In only one instance have I seen it remain limited to the first division of the fifth for years without either the second or third division becoming involved, and even in this case rubbing the chin would start the pain in the forehead. Practically always the neuralgia also involves the second or even all three divisions when the supra-orbital distribution is involved, though it is to be remembered that the pain in the second division of the fifth frequently, indeed generally, extends above the eyebrow and in front of the temple.

In all cases of persistent or recurrent pain, anæsthesia must be looked for. If it is present in trigeminal cases then either syphilitic neuritis, gumma or tumour is the cause.

Geniculate neuralgia, or neuralgia affecting the distribution of the sensory fibres contained in the seventh nerve, has been fully described by Ramsay Hunt and others, though some deny the association of the seventh nerve with such neuralgias. Transient pain around and behind the ear, lasting for two or three days, is a commonplace in association with the onset of facial palsy, and often precedes the motor paresis. Much rarer are instances of true herpes zoster affecting the auricle in association with facial palsy. The distribution of the herpetic rash is usually on the concha and antihelix, though it may be found behind the ear where the pinna joins the scalp, and also along the posterior wall of the external auditory meatus. Such a distribution of herpes I saw once in a medical man, without facial palsy, who for many weeks afterwards had excruciating paroxysmal neuralgia affecting the ear, front of the ear, and back of the lower jaw and neck. Pierce Clark and Taylor, of America, describe a case of chronic tic douloureux affecting this area which was cured by operation by trephining and dividing the pars intermedia Wrisbergi, or sensory root of the seventh nerve, intracranially.

Glossopharyngeal neuralgia.—A rare form of chronic paroxysmal neuralgia or tic douloureux may affect the glossopharyngeal nerve. In its paroxysmal suddenness of onset, and in the severity of pain, glossopharyngeal tic is identical with trigeminal tic, for which it may easily be mistaken. Distinguishing it, however, from the latter, the pain in glossopharyngeal tic starts in the throat, in the region of the tonsil and anterior pillar of the fauces. The pain radiates to the ear, and especially just in front of the ear, along the back of the mandible, and into the upper part of the neck. I have met with two only of these cases, both of which had lasted over ten years, one a man aged 40, the other an old lady of 87. As in trigeminal tic, the first onset of the pain may be furious and sudden, in the man's case the pain striking him in the throat as he opened his mouth to eat a sandwich. When I met with these cases, now ten years ago, I knew of no reference to glossopharyngeal neuralgia, and in case the pain might be an unusual form of trigeminal neuralgia, I injected the third division at the foramen ovale in each case, quite successfully as regards destroying this branch, but without influencing the recurrent neuralgia. I have since seen somewhat similar spasmodic pain, with intense hyperæsthesia of the side of the neck, in a case of recurrent epithelioma in the tonsil, following seven years after extirpation of a vocal cord for malignant growth. It was this case which convinced me of the identity of the glossopharyngeal tic in the two previous cases referred to, and I have lately seen a description by Sicard in France of three cases met with during the war of glossopharyngeal neuralgia, which were cured by surgical help by division of the nerve in the neck.

Diseases affecting Nerve Trunks.

Chronic pain in the distribution of the trigeminal nerve may be due to tumours or gummata involving the sensory root of the fifth nerve within the skull or one or more of its branches externally. Tumours in the ponto-cerebellar angle may irritate the sensory root of the fifth and simulate trigeminal neuralgia for years. The pain however, though variable, is less spasmodic and not brought on by light touches or movements of the face. The main point in distinguishing lesions of the trunk or main branches from trigeminal neuralgia is the appearance of diminished sensibility in the affected area. Motor palsy of the muscles of mastication also may be present. When these signs of gross damage to the fifth nerve are found, trigeminal neuralgia may confidently be excluded unless neurectomy or alcohol injection has

previously been done. Peculiarly distressing cases to deal with are the naso-pharyngeal growths, causing persistent pain and increasing anæsthesia of either the second or third divisions of the fifth. When the growth invades the zygomatic fossa and involves the third division, deafness of that ear is usually produced by involvement of the Eustachian tube. When the growth is more central and invades the spheno-maxillary fossa, besides involving the second division of the fifth, there is produced presently diplopia and later proptosis and fixation of the eyeball through extension into the back of the orbit. Beyond anodyne remedies I know nothing that relieves these cases. Operation seems useless; the growth is never circumscribed and always recurs. In malignant growths of the maxilla pain may be exceedingly troublesome, even after excision, due to recurrent growth. This pain may be completely relieved by a successful alcohol injection of the Gasserian ganglion. Similarly the agonizing pain due to carcinoma of the side of the tongue and lower jaw may be completely arrested by alcohol injection of the third division of the fifth at the foramen ovale.

Persistent trigeminal pain for weeks and months, non-spasmodic, and accompanied by diminished sensibility of that side of the face, but without the pressure signs above referred to of Eustachian deafness, proptosis, and diplopias, should suggest gumma as a probable cause. Even if the Wassermann reaction be negative, it is well always to give anti-syphilitic treatment a trial. I have met with several such cases of syphilitic trigeminal neuritis which cleared up completely under treatment by biniodide of mercury or salvarsan injections.

Rib Pressure on Brachial Plexus.

Chronic pain in the arm and neck, running down to the inside of the hand, in women between 20 and 30, will usually suggest cervical rib as a cause through pressure on the first dorsal nerve, as it rises to join the inner cord. Wasting of the musculature in the hand and diminished sensibility along the inner border of the forearm to the wrist render the diagnosis more certain. If a skiagram demonstrates a cervical rib, the position is clear. Many cases of rib pressure due to the first rib alone, in the absence of a cervical rib, have been recorded; I have had two such cases successfully relieved by operation within the last six months. In one of them the first rib caused a prominent hard swelling above the clavicle which it was difficult to believe was not a

cervical rib when the skiagram proved the absence of the latter. The diagnosis is therefore more complicated and difficult now that we must realize that with symptoms suggestive of cervical rib the skiagram may be normal.

Causalgia.

During the war the frequency of cases of persistent agonizing pain due to injuries of nerves, often slight, was most remarkable. The large majority of these cases involved the median or internal popliteal nerves; though I have seen it also in the distribution of the ulnar, long saphenous, external cutaneous of the thigh, and radial nerves.

In some cases the nerve injury was so slight that no demonstrable anæsthesia was present—only an intense hyperæsthesia, varying at times, and liable to be aroused into an intense spasm of pain by a sudden emotion, noise, or vibration or light touch, thereby reminding us somewhat of the onset of the paroxysms in trigeminal neuralgia. The pain is described as of a burning heat—hence the name "causalgia" (*καιω*, I burn), and bursting sensations in the fingers were common. The pain in many cases lasted for weeks and months, and in some it was necessary to produce nerve-blocking, in order to arrest the pain, by alcohol injection of the trunk of the nerve above the injury. Common enough during the war, I have seen only one such case in civil life where a man tore his thenar eminence in some cog wheels two years previously. The pain was limited to the ball of the thumb, and immediate and complete relief was obtained by injecting the median nerve with alcohol just above the wrist. The nerve can be easily found here, as it lies between the tendons of the flexor carpi radialis and palmaris longus, and pricked with a hypodermic needle and injected. No other operation is necessary for this.

Painful stump was another very common sequel of amputation in the war.

Brachial and Sciatic Perineuritis.

Sciatica I have already referred to, especially in association with lumbo-sacral fibrositis, either of a rheumatic, septic, or traumatic origin.

A very chronic and obstinate type I would like to refer to here in which the most noticeable sign on examination is the scoliosis, a lateral flexion of the spine away from the painful side, causing the hip on the affected side to stick out. The lower cervical spine may be as much as 5 in. to one side of the vertical median line when standing. This scoliosis disappears on sitting or lying. It may be a result of a local

lumbar fibrositis in the erector spinæ in the region of the fourth and fifth lumbar transverse processes. Occasionally a definite area of deep tenderness can be localized and it is sometimes possible to procure an immediate and dramatic relief by alcohol puncture of the spot. Short of that treatment these cases are very chronic and resistant to ordinary treatment, and three months' rest in bed may be necessary.

Brachial neuritis is seldom so chronic as some of the sciaticas, though the acute form is probably more painful, due I think to the structure of the cords of the brachial plexus being denser and firmer than the sciatic and thus admitting of less swelling in inflammation of the sheath. This point is easily demonstrated in the dead body by cutting down on the sciatic and brachial nerve trunks and injecting fluid through a hypodermic needle. The sciatic takes it easily and swells up in an egg shape, but it is difficult to force more than a few drops into the main brachial trunks.

Chronic pain about the scapula and arm may be due to cervical rib, or to neurofibrositis, as already stated. A common cause of pain referred from the shoulder down the arm, perhaps to the elbow, its maximum usually about the insertion of the deltoid, is adhesions in the shoulder-joint, and resulting from some slight injury such as strap hanging, being jerked by getting on or off a bus or tram, or by a slight fall.

These cases are frequently diagnosed as neuritis. The pain starts usually a day or two after the injury and is due to a synovitis, the subsequent adhesions causing pain with every movement or pressure on the joint. Wrenching under gas, with subsequent passive movements and massage, usually effect a cure, but some cases are too painful to tolerate the after treatment and partial fixation is permanent. To be distinguished from this condition is the fixation of shoulder from adhesions subsequent to immobilization during the acute stage of brachial neuritis. The history of severe pains and pins and needles down to the fingers generally makes this point clear, but the treatment by massage, passive movements, or even wrenching under gas is the same.

Disease of Posterior Spinal Roots or Root Ganglion.

Post-herpetic neuralgia is one of the most inveterate and difficult neuralgias we have to treat. Due in part to an inflammation lesion in the root ganglion, in some cases to a neuritis of the nerve trunk, and in others even to inflammatory changes in the grey matter of the posterior

horn in the spinal cord, the pain is constant and wearing, causing great depression. Rare under the age of 50, chronic neuralgia following an attack of shingles is increasingly common in older people. In some the pain may persist for months and then gradually disappear, but in others it may remain for years and has led to suicide. With the pain is usually a severe numbness and constriction, and the area of scarring is usually partially anæsthetic. Beyond local anodynes and mild tonic treatment I hesitate to suggest more active measures. Alcohol injections in spinal cases is, I believe, quite useless, and division of posterior roots after laminectomy is, I believe, not always successful. I should be glad to hear experiences of surgeons on this point.

Herpes of the trigeminal area is mostly limited to the ophthalmic division, often called frontal herpes. A common sequel is numbness, sensation of constriction and paræsthesiæ of formication, not really amounting to pain. Occasionally, however, in old people subsequent neuralgia in the anæsthetic area is very distressing and persistent. For this, alcohol injection of the Gasserian ganglion may give complete relief, and I have done this in three such cases, but it is necessary to produce total and lasting anæsthesia equal to that resulting from a gasserectomy.

Chronic pain in the limbs, usually the lower limbs, may result from posterior root sclerosis, subsequent to meningitic lesions. *Hæmatorachis* or intraspinal hæmorrhage in the lumbo-sacral region may result from heavy muscular effort. The effused blood is apt to clot around the roots of the cauda equina and cause chronic irritation and pain in one or both lower extremities, with wasting and loss of reflexes, and possibly diminished sensibility. Lumbar puncture will clear up the diagnosis, a straw-coloured cerebro-spinal fluid being drawn off even a year or more after the onset. In one such case I had laminectomy performed and a large purple clot 4 in. long surrounding the roots of the cauda equina removed, eighteen months after the injury.

Tabetic pains are too well known to need enlarging on here. Their treatment is perhaps less satisfactory. Mercurial friction improves some, intravenous salvarsan yet others, while for those who do not notably improve under salvarsan only I have seen considerable numbers benefit immensely as regards severity of pain by intraspinal injections of the serum taken from the patient's blood after intravenous salvarsan.

Usually there is a strong reaction about two hours after the injection of 50 to 55 c.c. of the serum, severe pains in the limbs coming on and lasting twelve to twenty hours, followed by more or less complete

relief, which may last for years. Presumably the injection of the serum sets up some congestive state in the posterior nerve roots which acts beneficially on the chronic syphilitic neuritis which is the source of the pain. Root sclerosis may be due to other causes, such as hæmorrhage, toxic degeneration in diabetes, and other causes of neuritis.

Proceeding centrally, we find chronic neuralgic pain resulting from intramedullary lesions affecting the fillet and thalamus. Usually the pain is constant, burning, and pins and needles in character, but occasionally it is paroxysmal and neuralgic. More than ten years ago I showed before the Neurological Section a man of over 60, who had suffered from an attack of thrombosis of one posterior inferior cerebellar artery. As a result of the thrombosis implicating the side of the medulla with the fillet and the descending or spinal root of the fifth nerve, there was analgesia of the left fifth area and of the right half of the body, excluding the face and the forehead. In the analgesic area of the left fifth he complained of constant neuralgic pain, which nothing appeared to relieve. It is perhaps difficult to understand how a simple sclerotic lesion can cause persistent neuralgia in the absence of any irritating focus, and this is the only instance of this posterior inferior cerebellar syndrome which I have seen with permanent neuralgia resulting, though this particular thrombosis does not appear to be very rare, as I have met with twenty or more cases. It has been suggested that the persistent burning pain and paræsthesiæ in lesions of the thalamus or fillet is due to the spontaneous unrestrained activity of these nuclear centres for sensation. Similar pain is met with in some cases of syringomyelia and syringobulbia, pain referred to analgesic areas or analgesia dolorosa. Intramedullary spinal tumours also are liable to cause a burning pain as an early symptom, which may precede for many months any more definite localizing signs.

Psychalgia.

Pain of mental origin is usually distinctive in character, such as the vertical pressure pain, or clavus hystericus of some neurasthenic headaches. A mental neuralgia may usually be distinguished from a true neuralgia of peripheral origin by its distribution not being anatomical in form and overlapping other nerve areas, and especially in crossing the middle line. A parson, aet. 63, for 2½ years has had pain in the right great toe. Of three surgeons who saw him, one operated for exostosis, the second excised the joint, the third told him he had a kink in his colon, but let him off with an abdominal belt and paraffin

internally, and advised him to get a tendon cut. In spite of all treatment (or because of it) his pain is now much worse. On inquiring into his history, I found he was the child of first cousins, and that insanity was very prevalent in his family. As I could see nothing wrong with his foot or leg, I have little doubt that this pain was an instance of psychalgia. Occasionally there may be difficulty in diagnosis, as in a Jewish patient whom I saw for pain on the left side of the cheek and forehead and nose, but crossing the nose as far as the inner canthus of the opposite eye. He was anxious for injection treatment, and as he had had much medicinal treatment without benefit, somewhat against my better judgment I injected his second division. Although dense anæsthesia resulted the pain was not improved, rather worse. I then hesitated between advising suggestion treatment and injection of the ganglion. He would not have the former and again against my better judgment I injected him, this time through the foramen ovale, and producing total and permanent fifth nerve anæsthesia. His eye and cornea fortunately gave no trouble, but he was now even more complaining of the pain, and he went to see another surgeon regarding gasserectomy, who, however, refused to do the operation when he found the fifth nerve area totally anæsthetic. Baulked in this he went to yet another surgeon who operated to remove his ganglion; and the last I heard of him was that he was still complaining violently of pain.

The moral of this tale is that it is unwise to attempt any form of surgical treatment for psychalgias; the pain is apt to get worse, or spread to another area, and once an operation or injection has been performed, it is most difficult for another practitioner to sift the real from the false and make a diagnosis. With the psychalgias may perhaps be included many of the *coccygodynias*, though perhaps in the majority of them there is a history of some local injury at the outset.

In conclusion, I must apologize for the discursive nature of my remarks as I have endeavoured to point to special types and difficulties which the combined talent here to-night will, I hope, throw light upon.

On Persistent Pain.

By Sir WILLIAM THORBURN, K.B.E., C.B., C.M.G., F.R.C.S.

THE treatment of a symptom rests always upon a more uncertain basis than does the treatment of a lesion. More especially is this the case with such a symptom as pain, which is not only purely subjective but the nature and intensity of which is not capable of measurement or even of definite expression.

Hence the estimation of the results of treatment requires considerable caution. On the one hand, such results may be due merely to suggestion, as was probably the case with alleged "cures" of tabetic pains by suspension or by stretching of the sciatic nerves—methods which I take to be now quite obsolete. On the other hand an operation which has certainly removed the pain to which it was directed—such as gasserectomy for trigeminal neuralgia—may be followed by the most disagreeable phenomena of "psychalgic" origin. Thus I have seen removal of the Gasserian ganglion followed by a functional hemianæsthesia, and in two typical cases of rib-pressure removed by operation the patients shortly developed abdominal troubles associated with a well defined movable kidney. As moreover many of the cases operated upon for persistent neuralgia are the victims of alcohol, morphia or other narcotic drugs, the picture is often obscured by such conditions, and again the judgment of operation results may be thereby rendered extremely difficult.

We are, therefore, on the safest ground when we are attacking a quite definite lesion, as in removing a source of pressure. We are less certain of results when dealing with root ganglia, whether spinal or cerebral: and we are on the most doubtful ground when dealing with diseases of obscure pathology such as are many of the neuralgias of the limbs.

With regard to pressure lesions, including those of the dome of the thorax, we are on very safe ground, and in nearly all, if not all, cases the removal of the cause will prove curative. The treatment thus resolves itself simply into the recognition of the cause. It is, however, possible, if not probable, that in cases of long standing there has been produced a local neuritis with cicatricial changes which may lead to permanent symptoms, although pain is seldom prominent among these.

With regard to pain due to external injury, the most typical

examples are those due to end-bulbs after amputation. The relief of sueh pain is generally immediate if the bulbs be excised, but in some cases little or no benefit will follow and even high resection of the affected nerves will fail to cure. In such cases there is again probably an extensive septic neuritis, as described especially by Corner, and for some of them posterior rhizotomy appears to offer the only hope of relief. A good deal of controversy has arisen as to the prevention of such end-bulbs, but my own opinion, founded upon a considerable experience of amputations dating back to a time when many were not aseptic, is that their essential cause is simply sepsis and that section of the nerves at a high level at the time of amputation is a sufficient prevention even in septic wounds. I have not practised any of the methods of occluding the cut ends of the nerves and I am not aware that their omission has given rise to trouble. Upon this point, however, I should greatly like to hear the experience of those who have worked in British hospitals during the war.

With regard to causalgia again the views of those who have had British war experience would be most welcome to me. So far as I can judge Sicard's method appears to be most popular, although I have no personal experience of it. It has, however, one fairly obvious limitation. When the injury lies near to the trunk the neuritis may well have extended so far that it is impossible to inject the nerve above it and it would then again appear reasonable to fall back upon rhizotomy. Similarly resection and suture may quite fail to cure and may leave rhizotomy as our only hope. (Platt found that of twenty cases treated by resection and suture sixteen were cured and one failed even after repetition of the operation, while of three treated by neurolysis only one was cured.)

Rhizotomy is however by no means always effective in this and other severe forms of neuralgia of the limbs. The analysis of Foerster and of various British surgeons given in the *British Journal of Surgery* yields fifty-nine cases of which twelve died or were not traced, while twenty were cured, and in twenty-seven there was either no relief or such relief was partial only. Of two cases of my own one was little if at all relieved, and it is interesting to note that this was a case of avulsion of the brachial plexus in which there was no open wound or septic infection, and in which it was obvious at the time of operation that the roots were matted together by a cicatrix, and that some cicatricial tissue extended into the spinal cord. Here we have a probable clue to the many failures, viz., an extension of disease beyond the posterior root-ganglia.

Before leaving the question of injuries of nerves, I should like to refer to the often very painful cicatrices of the scalp. Why these should cause much pain—sometimes ensuing long after the original injury—is not obvious, but I am strongly inclined to attribute it to attachment of the mobile scalp to the skull and I have certainly found that freeing of the scalp, with or without interposition of some material such as cargile membrane or aseptic wax between it and the bone, is followed by the relief of pain and of other nervous symptoms often associated with it.

Turning now to the brachial, sciatic and other neuralgias of more obscure pathology, these are of course widely treated by non-operative measures or by such measures as acupuncture, saline injections and "bloodless" stretching. Many of the cases recover as do many under the influence of rest alone, and I do not wish to dispute that all these methods have been followed by good results. But in the cases which come before me they have usually been tried and have failed. Hence perhaps I am strongly prejudiced in favour of a full exposure of the nerve. Such an exposure is free from risk and often reveals a definite lesion such as a source of pressure or a perineuritis with light adhesions. Neurolysis, doubtless associated with some stretching, is at least as likely to be curative as the more obscure attacks of apparently minor surgery, while it certainly provides a more complete and logical line of treatment. In many, but by no means all cases, it has given very good results.

Lastly, I must refer to ganglionic and tabetic pains.

As regards trigeminal neuralgia the various modern methods are so well known that it is unnecessary to do more than briefly refer to them. There are two great classes of operation. Schloesser's method of injection and Hutchinson's method of removing the two lower thirds of the ganglion have the merit that, so far as I know, they never imperil the eye, and in my own experience Hutchinson's operation has always been permanently curative. It would, however, be useless in the rare cases in which the ophthalmic division is involved. On the other hand, Haertel's method of injection, total ablation of the ganglion and resection of the sensory root have, to my knowledge, all caused loss of the eye even when performed by the most competent operators. Haertel's injection is the simplest and the safest as regards life, but appears to me the most uncertain and does not commend itself to one who likes to see what he is doing. Division of the posterior root—especially by Adson's technique—will probably supplant total gas-

serectomy, but so far I have always been satisfied with Hutchinson's method.

Post-herpetic neuralgia I have found surprisingly disappointing, but both my own and the recorded cases of root resection for this condition are too few for safe generalization at this time. Rhizotomy would appear to offer the best hopes of cure, but here again it may well be that changes have extended above the level of the root ganglia.

Tabetic pains are becoming increasingly rare, probably as a result of the better early treatment of syphilis. Foerster and Hey Groves have between them collected seventy cases with seven deaths, fifty cured or greatly improved and thirteen failures. Such results are well worth obtaining, especially as the death-rate is very much lower in the hands of surgeons accustomed to laminectomy, and there is also not a little evidence that spinal drainage, *per se*, is of value in locomotor ataxia. Moreover, several of the failures have been clearly due to a too limited resection of roots. Such resection, to be effective, should probably include at least the fourth, and eighth or ninth thoracic roots in cases of gastric crises and should generally be bilateral. I am quite satisfied that, if it be thoroughly carried out, we have in this operation one of real value which ought to be considered in every case of tabetic pain and which ought in the hands of experienced surgeons to be as safe as an exploratory laparotomy.

DISCUSSION.

Dr. GORDON HOLMES said that it was the teaching of English neurology that most diseases of the central nervous system which did not involve the meninges or extend to the posterior or other sensory roots ran a painless course. That that doctrine was true in the majority of cases there could be no doubt, but he was afraid it had been applied rather too widely. It was generally assumed that spinal diseases could produce pain only when the posterior roots were involved, but as a result of his war experience of gunshot injuries he thought it must be regarded as a possibility that a traumatic injury of the pain-conducting tracts within the spinal cord could produce pain, persisting for several weeks at least. It was not so rare as it was generally assumed to be for intramedullary lesions of the cord to produce pain. Perhaps the most interesting point from the theoretical side concerned the manner in which these pains occurred. It was an old hypothesis that the lesion irritated the pain-conducting fibres, and therefore gave origin to the pain which was peripherally referred or projected. He did not think that many of them were willing to accept that explanation now. He had found in certain gunshot injuries of the spine that not merely painful or uncomfortable sensations were

produced, but also an excessive sensation of what might be called pleasure on the affected parts of the body. The easiest working hypothesis in discussing the nature of the central pain was to assume the view put forward years ago by Long, that the pain-conducting fibres throughout the central nervous system were represented by chains of neurons broken up repeatedly in masses of grey matter, and that the pain was due to some structural or dynamic change in portions of grey matter in which the pain-conducting fibres were normally interrupted.

Dr. S. A. Kinnier Wilson said that if it was the case that the tic movement of a tic douloureux disappeared when the neuralgia was relieved—whether it was a tic or spasm was for the moment immaterial—it seemed as though certain inveterate cases of torticollis might be treatable by the relief of the accompanying occipital neuralgia by operation on the posterior root ganglia. Torticollis was a very interesting subject, and often cases of torticollis were particularly untreatable. He also referred to the description in Dr. Harris's paper of certain patients whose pains came on only at certain times of day (for example, a woman who had the pains from 12 to 5 o'clock each day). These cases presented very interesting problems from a theoretical point of view. The view he himself took was that such cases were essentially psychogenic. He did not believe that any factor except one of that nature could produce the recurrence of pain over the same period of hours every day. He added that he had used ionization successfully in several cases of post-herpetic neuralgia when other forms of treatment had been unsuccessful.

The President said that his own experience of posterior root section had been extremely disappointing, and on very few occasions had it been worth doing; especially was this true of doubtful cases of persistent pain of causalgic character. Every kind of surgical treatment for the relief of these pains had been tried and all had had a measure of success, but on the whole there was more disappointment than otherwise. Unless the nerve could be dealt with at a high level and at an early stage there was little likelihood of any great result from alcohol injections, resection of the nerve, or anything of the kind, and after the pain had persisted for years, then the posterior section itself had very little influence.

Dr. Wilfred Harris, in replying to the point raised by Dr. Wilson as to the periodicity of the pain, would not subscribe to the opinion that the periodicity meant psychalgia. Periodic pain was familiar to many of them in other connections. Moreover, the pain in this case had an anatomical distribution, and, contrary to the usual results in psychalgia, injection of the ganglion produced almost complete relief.

CLINICAL MEETING HELD AT THE HOSPITAL FOR PARALYSIS AND EPILEPSY, MAIDA VALE, DECEMBER 8, 1921.

Dr. WILFRED HARRIS, Vice-President, in the Chair.

(1) *Case of Division of Right Sciatic Nerve six years ago; Suture of Nerve six weeks later. Muscles show no wasting, but the Response to Faradism is diminished, and Voluntary Contraction is absent. The Calf Muscles are in spasm.*

By Dr. JAMES COLLIER.

W. H., aged 31, ex-soldier.

History.—Bullet wound severing right sciatic nerve in upper third of thigh July 30, 1915. Operation September 9, 1915. nerve found completely divided, ends sutured.

Discharged from hospital in June, 1916. Patient states there was wasting in right lower leg at this time. No treatment since, but patient always kept on his legs with the aid of a stick. By February, 1917, right leg had regained normal contour and measured practically the same as the left.

Admitted National Hospital, November 29, 1921.

Present condition.—Calf measurements at their thickest girths both 15 in. Muscles of right calf feel firmer than those of left. Constant fibrillary tremor of right calf muscles. Response of these and the fore-tibial muscles to faradism present with a strong current. No voluntary movement of right toes or at ankle, good movement at knee-joint. Sensibility to pin-prick is abolished from the lower third of the thigh downwards.

(2) *Post-encephalitic Lenticular Rigidity.*

By Dr. GRAINGER STEWART.

H. S., aged 9 years.

History.—Eighteen months ago patient had an illness during which he was in a sleepy condition for six weeks: could always be roused; both eyes turned inwards for two to three weeks; seemed unable to see for about a fortnight; had pain in back of head.

After drowsy state passed off, he began to have violent choreic movements of arms and head; legs slightly affected; right side worse than left; head turned to right. Movements lasted six months; as they gradually ceased, right side became weak.

Present condition.—Looks healthy. Face almost expressionless when at rest, quickly lighted up by smile.

Walks with body bent forward, tends to run. Cannot stand still without effort, but tends to fall backwards.

Mental condition: Very bright. Rather sly and precocious; often disobedient; sometimes sulky.

Signs: Pupils equal, react well to light, badly on convergence. External ocular movements full; deviation to left accompanied by nystagmus.

Fundi normal.

Hand-grip and arm movements slightly weaker on right side.

Resistance to passive movements of right upper and lower limbs.

Tendency to Parkinsonian posture.

Knee jerks and ankle jerks present.

Plantars probably flexor, but brisk voluntary extension movements usually occur.

Speech: Often high-pitched intonation—this is not constant and is probably due to habit.

(3) *Gummatous Spinal Meningitis.*

By Dr. A. FEILING.

F. D., female, aged 39, single, was admitted complaining of loss of power in the right shoulder.

Three years ago she had an attack of rheumatism with severe headache and pain in the tip of the right shoulder.

Two months ago pain in the right shoulder again.

Six weeks ago noticed difficulty in raising the arm above the head, and weakness of the arm increased rapidly.

On admission, October 5, 1921, pupils, discs and cranial nerves normal.

Well-marked muscular atrophy and weakness of muscles of the right shoulder, especially deltoid, spinati, biceps, and to a lesser extent of the pectorals, rhomboids and supinator longus muscles. No fibrillary twitching seen. No loss of sensation. Left arm not affected. Tendon reflexes in the right arm not obtained with the exception of a feeble triceps jerk. Only other abnormal signs found were absence of the abdominal reflexes and a double extensor plantar response.

Wassermann reaction in the blood strongly positive. Cerebrospinal fluid, Wassermann strongly positive. Cell count 40 per c.mm. Nonne-Apelt test for globulin positive. Slow but steady improvement is taking place under treatment.

(4) *Disseminated Sclerosis (with Chief Incidence on the Pons).*

By Dr. A. FEILING.

P. L., male, aged 31, complains of weakness of the left side of the face, loss of power in the right arm and right leg.

History and present condition.—August, 1916, fell from a motor lorry; no loss of consciousness. Next day felt "tingling" in the right arm and right leg; within twenty-four hours left facial paresis had appeared; three to four days later marked weakness of right arm and right leg; occasional severe

headaches, no vomiting. Two weeks later deafness in the left ear and left tinnitus. About this time precipitancy of micturition and occasional incontinence of both urine and fæces occurred: also loss of sexual power.

Condition has remained practically the same in last two years: at times the power seems to go from his right arm and leg, with cold feelings in the right side of the body. Occasional headaches; transitory diplopia.

On examination: Pallor of both optic discs. Nystagmus, especially on looking to the right. Diminution of sensation in and paresis of the left side of the face.

Well-marked right hemiplegia with spasticity of arm and leg: tendon reflexes on right side exaggerated; right plantar response extensor, left doubtful, possibly also extensor. All forms of sensation diminished in right arm and right leg.

Sphincters: "Precipitancy of micturition" well marked; no incontinence while in hospital.

Wassermann reaction negative in blood and cerebrospinal fluid (February, 1921).

(5) *Syringomyelia, with Predominantly Unilateral Signs.*

By Dr. A. FEILING.

R. A., male, aged 36, admitted to hospital complaining of loss of sensation in the left hand and arm and weakness of the left arm and left leg.

Two years ago blisters began to form on the left hand.

Eighteen months ago weakness left hand was noticed and the left leg occasionally gave way; loss of sensation in the left hand began at this time.

Constant sweating of the forehead has occurred during the last year. No pain.

On examination: Markedly excessive sweating of both sides of the forehead and on the left side of the face. On the left side the pupil is small, the palpebral fissure narrowed and the eyeball sunken.

Muscular atrophy is seen in the left hand and forearm, in the right shoulder girdle and slightly in the right hand and forearm. Much muscular weakness of left hand and forearm. Trophic lesion of left hand. Both legs are spastic, but especially the left.

Reflexes: Tendon reflexes in the arms are lost, with the exception of a feeble triceps jerk on the right side. Abdominals present on the right and absent on the left side. Knee jerks and ankle jerks exaggerated both sides; ankle clonus on the left side. The plantar response on both sides is extensor. There is prominent kypho-scoliosis.

On the left half of the body sensibility to light touch, heat and cold is diminished; sensibility to pain on the face, trunk and lower limb and abolished on the upper limb. Recognition of changes in passive position and of vibration (C_{128}) and appreciation of deep pressure is gravely disturbed in the left upper limb. There is no loss of sensibility on the right half of the body.

(6) *Involuntary Muscular Movements. Torsion Spasm.*

By Dr. A. FEILING.

A. W., aged $13\frac{1}{2}$ years, a Jewish boy, came under observation in July, 1921, for involuntary movements of the head and arms.

He had suffered from an attack of measles as a baby.

No history of any similar complaint nor of any nervous disease on either side of the family.

Symptoms were first noticed when the boy was between 4 and 5 years of age. He would then fall forwards off a chair in which he had been placed. The movements began gradually, first in the neck, and later in the arms, when the peculiar method of grasping an object was noticed. He has been in about the same state since the age of 8. Went to school and has always been mentally alert and bright. Speech normal except for interference caused by the movements.

A healthy boy without signs of disease in heart, lungs, or abdomen. Beyond the involuntary movements the only positive signs are: (1) A lateral nystagmus; (2) inco-ordination of the arms.

No rigidity; no actual paralysis; no loss of sensation; no alteration in the reflexes.

Wassermann reaction in blood is negative.

(7) *Double Ophthalmoplegia Externa (? Congenital) with Generalized Muscular Atrophy.*

By Dr. A. FEILING.

R. M., aged 13 years, male, was brought to the hospital for "weakness of the legs."

Previous history.—Sat up at 12 months, walked at 18 months, talked at 2 years. At age of $2\frac{1}{2}$ years, after an attack of measles, "went off his legs."

Chief complaint is of weakness of the legs, so that he becomes very rapidly tired after walking a short way and has to rest before being able to proceed.

On examination, a small undersized boy. He presents a condition of generalized lack of muscular development with marked weakness of all muscles, especially those of the trunk: thus he was unable to sit up in bed without assistance when he was first in hospital. In addition there is a condition of double partial external ophthalmoplegia, with partial ptosis and defective ocular movements in all directions.

There is slight nystagmus. Vision: right $\frac{6}{9}$, left $\frac{6}{6}$. Fundi normal. No alteration in reflexes; no loss of sensation.

Wassermann reaction negative in blood and cerebrospinal fluid.

(8) *Involuntary Movements, a Sequel of Encephalitis Lethargica.*
By Dr. D. McALPINE. (Dr. WILFRED HARRIS.)

Mrs. M., aged 34. In November, 1920, began to suffer from insomnia; on December 14 she saw double and the diplopia persisted for some days. On December 15 drowsiness developed, which later became marked; she slept day and night. Admitted to Maida Vale Hospital, December 23, under the care of Dr. Wilfred Harris.

Examination showed patient to be very drowsy, but she could be roused and answered questions rationally; speech slow and monotonous; no cranial nerve or ocular palsies: no diplopia; no myoclonic or other movements. During January, 1921, she improved. In March she became more drowsy and involuntary movements were first noticed; these have persisted with varying intensity ever since. She relapsed in June and developed an acute arthritis of the left shoulder-joint as a result of the violence of the movements. There was a further slight relapse in September when a divergent strabismus with loss of power of convergence and defective accommodation were noticed and this condition is still present. At no period has there been any evidence of involvement of cranial nerves or of pyramidal tract; the deep reflexes, sluggish at onset, are now brisk. Plantar responses have always been flexor. The involuntary movements which are now present are rhythmic and uniphasic, occurring about fifteen times to the minute, each spasm lasting about one second. They occur synchronously in the various parts affected. They vary slightly in intensity and regularity, but unless controlled medicinally they are extremely violent and painful and make the patient's life a misery to her.

She can inhibit them to a certain extent, but the next spasm is reinforced. They are increased by emotion and disappear entirely during sleep. Muscular tone in the limbs, in the intervals between the spasms, is not increased.

During an involuntary movement the chin is turned slightly to left; the occiput is retroflexed and approaches the right shoulder. At first, the angle of mouth on the left side was drawn downwards, but now there is usually no such movement. Left arm is internally rotated and pulled slightly backwards, extended at elbow and wrist; forearm pronated and fingers powerfully flexed into the palm. There is shrugging of right shoulder and slight abduction of whole of right arm. The trunk is flexed to the left. The left leg is slightly flexed and sometimes abducted at hip; the knee is powerfully extended; the feet and toes point downwards and the foot is slightly inverted. In the right leg there is slight contraction of hamstrings and dorsiflexion great toe.

Luminal alone of the hypnotics that have been tried seems to control the movements; the dose required is 6 gr. daily. This amount has been given for the last six months and, apart from slight paresis of accommodation, no toxic effects have been observed.

(9) *Involuntary Movements, Sequel of Encephalitis Lethargica.*
By Dr. D. McALPINE.

J. T., aged 52, became ill on December 9, 1919. Pain back of neck. Slept for two weeks; delirious. Afterwards found he could not read properly;

also had difficulty in speaking and weakness in left arm and left leg. In January, 1920, involuntary movements began which have persisted since; left shoulder first affected.

Pupils equal; react normally to light and accommodation. No ocular palsies. Speech slow and slurring. Cranial nerves normal. Slight loss of power left arm. Reflexes and sensation normal in upper and lower limbs. The involuntary movements affect chiefly left face and left arm. They are uniphasic and rhythmic, occurring about sixteen times to the minute. Each spasm lasts about one and a half seconds. They disappear during sleep. They occur at irregular intervals, two following each other often in quick succession; movements of arm sometimes occur without any accompanying movement in face. Peripheral stimulation and emotion increase the amplitude of the movements. In an involuntary movement the head is laterally flexed to left. The chin is depressed, the left angle of mouth is drawn outwards and left cheek sometimes sucked in. The left shoulder is slightly raised, the whole arm being pulled backwards, with the elbow extended, the forearm pronated, and the wrist slightly flexed. The index finger is either flexed or extended, but the middle finger constantly flexed and the thumb abducted, Movements in the arm are often not so complete as those described above. There is occasionally slight contraction of the extensors of left knee. The involuntary movements do not involve the right arm or right leg.

The patient has benefited slightly by the administration of hyoscine; small doses only of luminal have been tried without apparent benefit.

The main features in these two cases are strikingly similar; the nature of the movements seems to indicate a "release" phenomenon; the site of the lesion or lesions is problematical; the absence of rigidity in the interval between the tonic spasms practically excludes a lesion of the corpus striatum. which combined with the fact of absence of cranial nerve involvement seems to point to a lesion in the subthalamic region.

(10) *Case of Cerebrospinal Syphilis.*

By Dr. H. MAUDSLEY. (Dr. FEILING.)

A. W., aged 56, labourer. Married, with two children alive and healthy; wife has had six miscarriages.

He was infected with syphilis thirty years ago. Has always felt well up to onset of present condition. Fairly rapid onset eight months ago of weakness of left hand and forearm. First noticed that he was unable to approximate thumb to his fingers on the left side—the disability gradually progressed with weakness of the hand-grip and wasting of the muscles of the hand and partial loss of power in the forearm and upper arm.

On examination his pupils are found to be equal in size, but slightly irregular in outline; they react very sluggishly to light, but briskly to accommodation. Other cranial nerves are normal.

There is evident flattening of the thenar and hypothenar eminences of his left hand together with some wasting of interossei. He can flex his inner two digits, but is unable fully to flex his thumb or outer two fingers. His power of extension of wrist is fairly good, but he cannot fully extend the distal phalanges. There is some loss of pronation and supination and flexion of left forearm and slight general weakness of the upper arm. There is also a certain amount of wasting in forearm and upper arm. The muscles of the wrist and hand are hypotonic. The power of the right upper limb is good. There is no weakness of his lower limbs.

Upper limb reflexes on both sides are fairly active. Knee-jerks present, left slightly greater than right. Ankle-jerks: left is just present, right absent. Abdominal reflexes are absent left side, just elicited on right side.

Plantars: Double extensor. No clonus and no spasticity of lower limbs.

Sensation: Apart from a slight defect in the recognition of vibrations on his lower limbs, there is no sensory loss. There is no unsteadiness when he stands with his eyes closed.

Wassermann test is strongly positive in blood and cerebrospinal fluid. Cell count in cerebrospinal fluid 22 per cm. Nonne-Apelt test strongly positive.

(11) *A Case of Pain due to a Central Lesion of the Spinal Cord, probably Syringomyelia.*

By Dr. SYMONDS.

The patient, Mr. L. H., aged 45, complains of continuous pain in the left shoulder and arm. He gives the following history:—

Antecedents: He has been employed in the manufacture of water-colours for twenty years. He has had no serious illnesses since childhood, and denies venereal disease. Nineteen years ago he accidentally cut the palmar tendons of his right hand. Thirteen years ago he had an abscess in his left thigh. He has been married twenty-three years, and has three healthy children.

Present illness: About nine years ago he first noticed while at work a feeling of roughness in the skin covering the thumb and first finger of the left hand. "It did not matter what he touched, it felt like a bit of sandpaper." This feeling of roughness later became continuous, being present whether he touched things or not, and gradually spread upwards until about two and a half years ago he experienced the same sensation all the way up his arm, across the left half of the chest above the nipple and over the left shoulder.

Four years ago he burned his left thumb with a poker and a blister was produced without any sensation of heat or pain. This experience has since been repeated on several occasions. In August, 1921, while in hospital, letting his left hand rest upon a hot-water pipe he suffered a severe burn upon the dorsal surface of the forefinger, this being brought to his notice by the appearance of a blister only.

Three years ago he was first aware of some weakness of the left hand

and wrist. This has steadily progressed until he is now unable to use his left hand for handling objects or for doing up the buttons of his clothes. On this account he was obliged to give up work two and a half years ago.

Eighteen months ago he first noticed involuntary jerking movements of the left arm, beginning in the muscles of the shoulder.

Two and a half years ago he began to have pain. At first this was experienced only in the left shoulder, being described as "a tight drawing feeling." This has increased in severity, and as time went on would sometimes shoot down the arm into the limb and first finger. Latterly it has spread to the hand and other fingers.

Two years ago he became aware that when he touched the skin over the lower part of the left face it would cause a shooting pain "like electricity" to travel down the left arm into the finger-tips. He discovered for himself that a light touch or flick of the skin with the finger is most likely to give rise to this painful sensation, or even a touch with the tip of the tongue upon the skin at the angle of the mouth. Continuous pressure is not followed by the same result. He therefore shaves that side of his face with a continued steady pressure of the razor instead of a series of short strokes. He is quite definite in his statement that the skin area from which this reflex pain can be elicited extends exactly to the midline of the face.

Examination of central nervous system: The patient does not give a very clear history, but is quite a fair witness to his sensations.

Special senses: Examination reveals no abnormality.

Cranial nerves: The left pupil is slightly larger than the right, and dilates more rapidly after stimulation with light. Otherwise there is no abnormality in the domain of the cranial nerves, except the sensory changes in the trigeminal field described below (see accompanying charts).

Sensations: Light touch, especially a flick with the finger, applied to the skin of the lower part of the left cheek gives rise to a sensation described by the patient as "an exceedingly painful electric shock," shooting down through neck and shoulder into the left arm and affecting especially the limb and forefinger. The threshold for the reflex is lowest over the area supplied by the third division of the left trigeminal, and extends exactly as far as the midline in front. The reflex can also be elicited from the lower part of the area supplied by the second division of the trigeminal on this side, but the threshold here is higher.

To cotton wool there is loss of sensation over the left shoulder, and diminution of sensibility not amounting to complete loss over the left arm.

To pin-prick there is complete insensibility of left arm and shoulder, and of the trunk between the second dorsal and ninth dorsal levels. The area of analgesia on the left side extends upwards to include the left side of the back of the head and neck. There is also a subjective diminution to pin-prick over the left side of the face as compared with the right. There is partial analgesia to pin-prick of the right arm affecting mainly the posterior surface and ulnar side. To heat and cold the cutaneous loss follows much the same distribution as that for pin-prick.

Motor system: At rest there are apparent certain involuntary movements mainly confined to the left upper limb. These consist of irregular clonic spasms affecting either single muscles or their fasciculi.

The muscles chiefly affected are the pectoralis major, triceps, biceps, latissimus dorsi, long flexors and extensors of wrists and fingers. On the right side also there are occasional twitchings of the pectoralis major.

There is some wasting of the intrinsic muscles of the left hand and general weakness of the left upper limb affecting mainly the power of hand and finger movements.

Reflexes.—Upper limbs: Tendon jerks on the right are brisk: on the left absent.

Abdominal: All present; the right perhaps a shade brisker than on the left.

Lower limbs: Knee and ankle jerks are brisk, greater on the right than on the left. The plantar reflex on the right is flexor; on the left indefinite. The sphincters and gait are normal.

The Wassermann reaction is negative in blood and cerebrospinal fluid. The cell and protein content of cerebro-spinal fluid are normal. The X-ray plates of the cervical and upper dorsal vertebræ show no abnormality.

This would appear to be a slowly progressive lesion of the central portion of the cord in the upper dorsal and cervical regions, probably a syringomyelia, in which for the past two and a half years continuous pain of a severe character has been the most prominent feature.

I would draw particular attention to the pain of reflex origin produced in the neck, shoulder and arm of the left side by stimulation of the lower trigeminal skin field on the same side of the body.

Notices of Recent Publications.

Anatomie du Système Nerveux. By Cornelis Winkler. Première partie, 1918, pp. 435. Deuxième partie, 1921, pp. 373.

The appearance of the second volume of this anatomy of the nervous system enables us to review the work as a whole; and while we regret that it is not yet complete, and look forward with interest to the appearance of a third volume, there is much in those before us to call for appreciation and comment.

The work is conceived on a large scale, and is intended to give a complete description of the anatomy of the nervous system from the terminal sense organs to the cortex. Nor does it stop there, but it traces the reflex paths along the association fibres of the cerebrum to the efferent tracts of the cord. There for the time being we leave them, but in the promised third volume a fuller description of these efferent paths and their effector organs, as well as of the cerebellum, will, no doubt, be given. The author does not confine himself to human anatomy. For, indeed, an anatomy dealing only with the human nervous system must necessarily be very incomplete, as little is definitely known of many of the tracts and systems in the human nervous system, of which the counterparts have been studied with considerable detail in the lower animals: and it is a mistake too often made in textbooks of anatomy to assume that what is true for the dog and cat must necessarily be true for man. Winkler avoids this error by giving along with descriptions of experimental work the clinico-pathological findings which correlate it with human anatomy. Much of his own work is thus presented to us, and with it we are glad to find numerous references to the work of English neurologists. In many places his observations have led him to conclusions which are not those generally accepted. Thus he considers that all the dorsal roots conducting sensory impulses from the skin enter by Lissauer's zone and end in the substantia gelatinosa of the segment in which they enter the cord. With regard to the descending tracts in the dorsal columns of the cord he considers that the "comma tract" of Schultze certainly, and the long descending tract of Hoche probably, are made up of exogenous fibres, and are, in fact, the prolongations of the descending branches of the dorsal roots. Again, the mesencephalic root of the trigeminal nerve appears to him to be a nerve in the autonomic system of the trigeminal, trochlear and oculomotor nerves. This is in opposition to the teaching of Ariëns Kappers, who considers it to be a part of the sensory root, the ganglion cells of which have not migrated from their original position within the cord. Such instances could be multiplied and

serve to show, what is indeed evident throughout the work, that the author relies chiefly on his own observations in forming conclusions on disputed points of anatomy. But in most instances where his views differ from those of others the reasons for this divergence are fully exposed, and the grounds on which the older views were based fairly stated. The reader is thus presented with data on which to form his own judgment.

The book follows a somewhat novel method of arrangement in that it traces tracts and systems according to their functions rather than their situation. This, though logical, does not always lead to clarity or brevity of exposition. In the first volume the olfactory and optic nerves are first described, along with all their connections with the brain-stem and cortex. Then follows a full description of the peripheral nerves subserving cutaneous sensibility, to which is added a full description of the spinal cord and its reflex paths, and of the connections of the spinal afferent paths with the brain-stem, thalamus and cortex. To this is appended a fairly short description, amounting to only twenty-five pages, of the longer efferent paths of the brain-stem and cord. In this connection he is very guarded as to the importance of the part played in human physiology by the efferent tracts arising in the brain-stem, and says "It is not yet proved, though it seems probable, that there are in the ventral and lateral pyramidal tracts fibres coming from the corpus striatum." This volume closes with two chapters of descriptive anatomy of the brain-stem, and one on the nervous apparatus of taste.

The second volume deals exclusively with the fifth and eighth pair of cranial nerves and their central connections. Of the 260 pages devoted to the eighth nerve, a large number deal with the recent work of Magnus and de Kleyn on the vestibular nerve. Following these authors Winkler provisionally divides the eighth nerve into three main components: (*a*) the cochlear nerve; (*b*) the fibres derived from the *maculæ acusticæ*, of which the part derived from the saccule runs with the cochlear division, and that from the utricle with the vestibular division; and (*c*) the fibres coming from the semicircular canals and ampullæ.

The volumes are well and profusely illustrated. The method of presentation is probably the best for those who, having studied the gross anatomy of the brain and cord, wish to learn their structure in relation to their function. They are emphatically books for the study rather than for the laboratory, and are much more suited for continuous reading than for reference.

J. G. Greenfield.

Les Tumeurs du Cerveau. By Viggo Christiansen. Pp. 337. Paris: Masson. 1921.

This book by a Danish author is, fortunately, published in a French translation, and in this form is a very useful addition to neurological literature. Following the modern fashion it takes the form of a series of clinical lectures. It is written in a simple and clear style and leads the reader in ordered sequence from those tumours which present such simple and

unequivocal clinical signs as hemiplegia and aphasia to those in which the signs are more complex and the localization more difficult.

It should therefore be especially valuable to the student of neurology who has not the opportunities given by a large clinic of seeing a great variety of cerebral tumours.

In the earlier chapters the author often seems to take a case of cerebral tumour as a peg on which to hang a general discussion of the function of various parts of the brain cortex, so that the reader is introduced to a wider survey of neurology than if the lectures were strictly confined to the matter in hand. Similarly in dealing with tumours of the base of the brain the author introduces cases of *migraine ophthalmoplégique* and of myasthenia gravis to illustrate the differential diagnosis. Tumours of the hypophysis and the infundibulum, and of the acoustic nerve, are thoroughly discussed from the standpoint of diagnosis. With regard to the latter the author emphasizes the importance of early diagnosis at a stage when the tumour is small enough to be completely removed by operation. He lays stress in the diagnosis of these tumours on a history of vertigo or tinnitus as an early symptom, on the early appearance of papillœdema, and on the presence of an increase of albumin in the cerebrospinal fluid.

In the last chapter but one on "Uncertain Diagnosis" he discusses the reasons why, in many cases, localization of a cerebral tumour may be impossible, and gives details of illustrative cases. The last chapter on "Surgical Treatment" is a lame finish to an excellent book, but like the previous one it shows the author's mental honesty. His own operated cases numbered thirty-three, and of these eleven only had survived up to the time of the lectures. Of these nine were tumours or cysts in the supratentorial region of the cranial cavity, while the other two were tumours of the acoustic nerve partially removed by the translabyrinthine route.

The book is one which will be read with interest by the experienced neurologist, and with advantage by the student of neurology, although it scarcely fills the rôle of a book of reference.

La Dégénérescence Hépato-lenticulaire. By H. C. HALL (of Copenhagen). Pp. 358. Paris: Masson. 1921.

This monograph deals with the diseases in which degeneration of the lenticular nucleus is constantly associated with cirrhosis of the liver. The name hepato-lenticular degeneration thus covers not only progressive lenticular degeneration, but also the pseudo-sclerosis of Westfall and Strümpell, and certain cases of torsion spasm. The author considers that these three diseases are closely related; and, while typical cases are clinically distinct, there are others in which it is not easy to make an exact differential diagnosis.

Progressive lenticular degeneration and pseudo-sclerosis are shown to have certain clinical features in common in addition to those due to disorder of motor function. The most important of these is a peculiar form of corneal

pigmentation due to the deposit of pigment in the outer part of the membrane of Descemet. It is much more commonly found in cases of pseudo-sclerosis than in those of progressive lenticular degeneration, and, so far as is known, does not occur in any other form of disease. Microchemical examination of this pigment proves that it is neither melanin nor lipochrome, nor yet either of the ordinary blood-derived pigments, hæmosiderin and hæmatoidin. On the other hand it corresponds in many particulars to the pigment produced by the activity of the plasmodium of malaria. It responds also to some of the chemical tests for the pigment of argyrism, but Hall considers that it is very improbable that it is derived from silver.

Another clinical feature common to the two diseases is the tendency to alimentary glycosuria, and the constancy, in the cases which have been tested, of alimentary lævulosuria. This tendency is, no doubt, associated with the depraved function of the liver. The nature of the cirrhosis appears to be identical in both diseases, although it has been variously interpreted. Some authors consider it due to faulty development of the hepatic lobules, while others attribute it to some enterogenous toxin. Hall thinks that it is a true cirrhosis, although it differs from the common cirrhosis of Laennec in being much more vascular, and, in consequence, having very little tendency to cause ascites.

He deals at some length with the familial nature of the disease. His own material consists of seven cases, only one of which was followed to autopsy. These seven cases were derived from three families, two from one and four from another. The latter family is of great interest inasmuch as the cases occurred in two generations, with a common male ancestor—two sons of a daughter by a first marriage and a son and daughter by a second marriage being affected. From this he argues that the disease is probably due to the fusion of two hereditary trends, one of which occurs very rarely and shows itself as a dominant, whereas the other occurs fairly commonly, and may be either dominant or recessive. In the instance quoted above the first hereditary trend would be present in the father, and the second in his second wife and the husband of the daughter of the first marriage.

The author deals fully with the pathology of the cerebral degeneration, not only detailing his own findings but giving a complete résumé of those of other workers. From this it appears that the degeneration is by no means confined to the lenticular nucleus, although it has its greatest intensity there. In pseudo-sclerosis particularly changes, especially of the nature of glial overgrowth with the formation of abnormal types of glial cell, were found in parts as distant as the cortex and the pons.

He deals somewhat scantily with the theories as to the physiological basis of the motor symptoms. In this he is probably wise, as our knowledge of the physiology of the corpus striatum still rests on somewhat insecure foundations.

The book forms a valuable addition to neurological literature in that it presents in readable form a full summary of work which up to now has been scattered in many lengthy papers in various languages. And the author's wise judgment and critical acumen add greatly to its value.

The Psycho-analytic Study of the Family. By J. C. Flügel, B.A. (The International Psycho-analytical Library No. 3). Pp. 259. London, Vienna, New York: International Psycho-analytical Press. 1921.

"A person whose instincts and impulses are co-ordinated sufficiently to maintain, as regards all the leading aspects of life, a relatively harmonious functioning of the whole personality can preserve mental health in circumstances under which a less integrated mind would fail." One of the most potent influences to which a human being is exposed from the cradle to the grave is family life. The object of this book is to study this factor in its various forms as it appears in abnormal psychology. Primitive emotions, conflict, the growth of individual personality, love and hate, are all examined from this point of view. The effect of early family influences on social development, religion, the attitude of parents to children and the various forms which may be assumed by sexual desire are described according to the teaching of Freud and his followers; the views of Jung receive due attention, though they are not accepted by the author. The book contains no original investigations, but is a useful compendium of the theories of the psycho-analytical school on the diverse manifestations produced by family life in the psychology of the individual.

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